VOL. CLIII, No. 4 APRIL, 1917 No. 541

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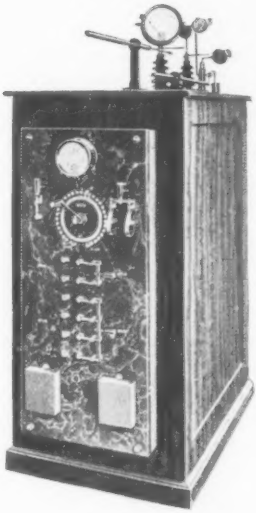

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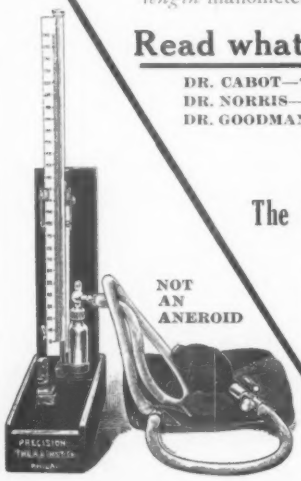

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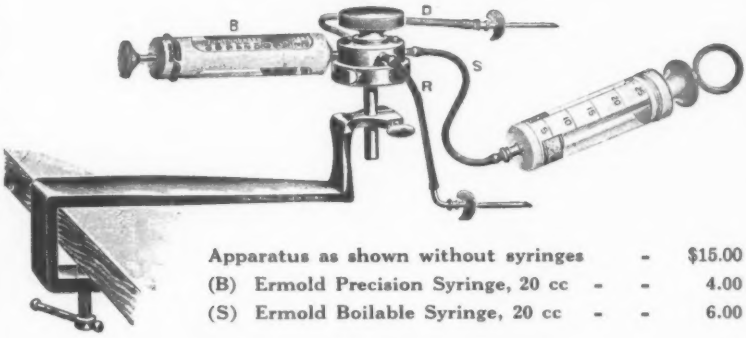

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ORIGINAL ARTICLES

THE DISTURBANCE OF THE LAW OF CONTRARY INNERVATION AS A PATHOGENETIC FACTOR IN THE DISEASES OF THE BILE DUCTS AND THE GALL-BLADDER.

By S. J. Meltzer, M.D., LL.D.,

NEW YORK.

(From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research, New York.)

The gall-bladder has a definite place in the physiological organization of the animal body. Like the urinary bladder it presents an ingenious but simple mechanical device, by means of which a continuous glandular secretion is transformed into a discontinuous elimination. The value of such a device is easy to see in the case of the urinary bladder; without its interpolation between the ureter and the urethra the continuously secreted urine would most unpleasantly drip continually from the body. An analysis of many other excretory processes in the body would readily disclose the presence of such a transformatory device in their mechanism, but we shall confine our present discussion to the secretion of the bile and the role of the gall-bladder.

While the secretion of the bile into the biliary capillaries is a continuous process the discharge of the bile through the common duct into the duodenum occurs only periodically. During the intervals between the periods of evacuation the bile is stored away in the then practically quiescent gall-bladder. We know a few facts which make such an arrangement appear to be advantageous

and physiologically desirable. It is an established fact that bile salts as well as pigment are reabsorbed from the intestines, to be stored up again in the gall-bladder. It is quite probable that the presence of bile in the intestines, at a time when it is not needed for a specific digestive action, is liable to exert some injurious influence. Its periodical storage in the gall-bladder presents therefore a protective functional advantage. Furthermore, the bile in the gall-bladder is a great deal more concentrated than that which is present in the system of biliary ducts. When therefore the intestinal digestion is in that state of digestion which requires the activity of the bile the intestine receives it by the aid of the gall-bladder at once in a concentrated form. The gall-bladder therefore is on the one hand the means of providing the intestines with bile in a concentrated form when it is needed for digestion, and on the other hand it may be the means of protecting the intestines or some of its function when the presence of bile might be injurious to them.

While we thus see that the periodical discharge of the bile into the duodenum and its storing up in the gall-bladder is a physiological and advantageous process, we ought to recognize that it harbors a pathological element, and that is stasis. Moderate continuous movements and exchange of composition of the body's material is conducive to a physiological healthy state of life; abnormal retardation and stasis lead to pathological disorganizations. As far as the gall-bladder is concerned we have to bear the following in mind: Bile even under normal conditions seems to contain often living pathogenic organisms. They may be brought there in various ways: By entering the common duct directly from the duodenum; by passing up from the intestines through the portal vein, and possibly also by elimination directly from the systemic circulation, because, according to Adami and several French observers, the circulation even in health contained such pathogenic organisms as the colon bacillus. As to disease, there is no doubt that during infection the systemic circulation is invaded by pathogenic microbes. For typhoid it is fairly well established that in nearly every case the blood harbors typhoid bacilli during the entire course of the disease. Nevertheless, even in typhoid, cholangeitis, and cholecystitis are comparatively infrequent complications. What prevents their frequent occurrence? Two elements are of importance in preventing the development of inflammatory processes in the biliary system, though even the bile contains living pathogenic organisms. One is the maintenance of normal vitality of the mucous membranes of the gall-bladder and the bile ducts; microörganisms prefer to settle and grow on tissues which lost their resistence. The other safeguarding factor is the absence of an abnormally prolonged stasis of the bile in the various parts of the biliary system. Bile stasis is of primary importance as a pathogenetic factor in biliary

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diseases. Stasis in itself may lead to a lessening of the vitality of the mucous membrane, and both together are then liable to make the mucous membrane of the biliary system the seat of inflammatory processes of various degrees and kinds: Catarrhal, suppurative, phlegmonous, membranous, or gangrenous. Furthermore, the inflammatory processes, when not of a stormy and fatal character, lay the foundation for the development of cholelithiasis. Besides, stasis alone, even without the intermediary action of inflammatory processes, favors the development of biliary calculi

We thus see that the physiological quiescence of the gall-bladder harbors a pathogenetic element. However, the periods of rest of the gall-bladder, as they exist in normal conditions, give apparently no rise to pathological phenomena. These set in, when the stasis of the bile is abnormally prolonged, of a chronic or a recurrent nature. What causes such abnormally prolonged conditions of stasis?

The data which pathology presents us are derived chiefly from observations made postmortem or in surgical operations. The observed facts, briefly enumerated, are as follows: When the gall-bladder or the bile ducts are in a state of inflammation the mucous membranes are red, swollen, and the lumina of the ducts, which are thus narrowed, contain in addition some mucus which increases the difficulty for the bile to pass out of the biliary system. In some instances Vater's papilla is even found to be completely closed by a plug of mucus. These conditions undoubtedly are capable of producing bile stasis, of diminishing or completely stopping the discharge of bile into the duodenum. But this stasis is here only a secondary phenomenon, it is merely a result of inflammatory processes, processes which, moreover, are themselves mere reactions, that is, they are themselves mere secondary phenomena brought about by some primary cause. Is there a form of stasis which in itself may be the chief, or a contributory cause of some inflammatory processes in the biliary system? Or, to put the question in other words, may not conditions happen which are capable of converting the periodically occurring physiological quiescence of the gall-bladder into an abnormally prolonged quiescence and thus converting the physiological storage of the bile into a pathological stasis? That is the central point of the problem I wish to discuss in this paper.

But before this question in pathology can be answered, there is a physiological question to be discussed and answered. What is the nature of the mechanism which in normal conditions causes the stream of bile at one time to be emptied into the duodenum and at another time to be diverted into the gall-bladder? This question was hardly ever taken into consideration when dealing with the pathogenesis of the diseases of the gall-bladder.

I have previously compared the mechanism of the gall-bladder with that of the urinary bladder. Of the latter we know that the

collection and evacuation of the urine is controlled by the antagonistic activities of two muscles of the bladder. During the accumulation of urine in the bladder the detrusor muscle is relaxed and the sphincter is tonically contracted, while the reverse takes place during evacuation, the detrusor contracts, and simultaneously the tonus of the sphincter becomes inhibited.

I could now apply directly the phenomenon of crossed innervation which we find in control of the activities of the bladder, without much discussion to the mechanism which dominates the activities of the gall-bladder. But I prefer to discuss here the phenomenon of antagonism from a broader point of view. The law of contrary innervation is manifest in all functions of the animal body, and I believe that a *disturbance of this law is a factor of more or less importance in the pathogenesis of many disorders and diseases of the animal body*. A better appreciation of this law, which can be brought about only by an insistent discussion of it, will render a service to pathology and clinical medicine. Thirty odd years ago I directed attention to the general principle that impulses inhibiting the action of antagonists must be an integral part of any kind of a movement in the animal body. For instance, simultaneously with each contraction of the extensors an inhibition of any form of contraction of the flexors, and *vice versa*, must take place. Otherwise, normal locomotion, for instance, would be practically impossible. I have demonstrated then, and recently we have shown it again, that this principle is an integral part of the respiratory mechanism. A few years before my general statement was made, we discovered that this arrangement is strikingly present in the mechanism of deglutition: The contraction of the lower parts of the esophagus and of the cardia become inhibited as soon as the upper end of the canal of deglutition begins to contract. Bayliss and Starling, Joseph and Meltzer, and Cannon and his coworkers reported observations of a similar reaction in several other parts of the digestive canal. Bayliss and Starling found that local stimulation of some segment of the intestines causes a contraction above and inhibition of the intestines below the stimulated part. They called it then the law of the intestines. Evidently this is not merely the law of the intestines but a part of a general law. Sherrington studied brilliantly this law, especially in the relations of the antagonistic muscles of the lower extremities. These relations exist there in a mutual way, and Sherrington termed them the reciprocal innervation. On account of the distinguished position of the investigator and the brilliancy of his work, physiologists are more familiar with this term and are apt to employ it in their writings. It is, however, evident that this is inappropriate to cover all phenomena manifestly belonging to this category. There is, for instance, no such "reciprocal" innervations in the physiological peristaltic movements of all parts of the digestive canal; there is

always only an inhibitory impulse for the caudal part of the intestine simultaneously with a contraction in the cephalic end of it. I therefore designated many years ago all these relations by the more general term of the *law of contrary* innervation.

I wish to add that many years ago I defined intestinal and gastric colic as being due to a disturbance of the law of contrary innervation—namely, that the caudal end remains contracted and stationary while the cephalic end contracts and progresses forward. The volume between the two contracted limits becomes gradually smaller while the contents of this volume remains practically unchanged; hence the increasing tension which leads to the unbearable colicky pain. Nothnagel, who previously went on record for another explanation of the origin of colic, admitted that some forms of colic may indeed have their origin in the described disturbance.

Let us now turn our attention to the application of the law of contrary innervation to the mutual relations of the evacuation of the bile through the common duct into the duodenum and to its storage into the gall-bladder. I dwelt on this subject some fourteen years ago in a paper on "Inhibition." We know that the gall-bladder is well provided with muscle fibers, the contraction of which will free the gall-bladder from its fluid contents. As to the termination of the common duct, Oddi described some thirty years ago that the papilla of Vater is provided with circular muscle fibers which by their contraction close up the common duct. This contraction is strong enough to resist a pressure much higher than that which usually obtains in the bile ducts. It is clear that the muscle fibers of the gall-bladder and those of the papilla are antagonists. Applying our knowledge of the mechanism of the urinary bladder, and more generally the law of contrary innervation, the physiological mechanism of bile storage and bile discharge is quite simple. During the storage the muscle fibers in the papilla are contracted and those of the gall-bladder are inhibited; during the discharge the gall-bladder contracts and Oddi's muscle is relaxed; the bile has then no other way out but into the duodenum. The innervation of the concerned parts, although not yet settled in its details, is very interesting. According to Doyon, stimulation of the peripheral end of the splanchnic nerves causes simultaneously a contraction of the gall-bladder and an inhibition of the tonus of Oddi's muscle. The vagus, on the other hand, seems to contain motor fibers for the sphincter of the common duct and inhibitory nerve fibers for the gall-bladder. Furthermore, also the afferent innervations show the character of contrary innervation. For instance, stimulation of the central end of the vagus causes simultaneously a contraction of the gall-bladder and an inhibition of the sphincter muscle.

While the physiological muscular and nervous mechanism of bile storage and bile discharge is thus satisfactorily explained the

question presents itself: What are the causes which bring about either of the two actions? We compared the mechanism of the gall-bladder with that of the urinary bladder. But the latter is to a large degree under the management of the will, and the sensations of fulness and other sensory stimuli bring the condition of the bladder to the attention of our consciousness, which by means of the will sets the required part of the mechanism into action. The processes of storing of the bile or of emptying it never comes to our consciousness and are never managed by our will. What does then manage their proper activity? We must admit that we do not yet know a great deal about it. It is probable that certain conditions and certain substances exert a selective action upon the reciprocal reflexes. It is interesting to quote here some newer instructive statements. One comes from Pavlov's school. Bruno stated that no bile appears in the duodenum as long as the stomach is empty. When a meal is taken the entrance of chyme into the duodenum gives the signal to an ejection of bile from the common duct. Most interesting are the recent studies of Rost. He first established the fact that after cholecystotomy the escape of bile through the papilla of Vater is indeed continuous, which in normal animals it is always a discontinuous one, depending upon the entrance of food into the duodenum. He found, further, the instructive fact that injection of peptone or albumosis through a duodenal fistula in a normal dog causes immediately a discharge of bile from the common duct, and he has proved that this takes place by a reflex act which causes a contraction of the gall-bladder and simultaneously a relaxation of the sphincter of the common duct.

From the foregoing it seems quite safely established that the physiological discontinuous character of the flow of bile into the duodenum is regulated by a reflex mechanism, dominated by the law of contrary innervation; that the integrity of the gall-bladder is an important part in this reflex mechanism; that the discharge of bile can be greatly curtailed by the absence or a restriction of the discharge of chyme from the stomach into the duodenum, and that the discharge of bile through the papilla of Vater into the duodenum is greatly enhanced by the presence in the lumen of the latter of peptone or albumosis.

We return now to our original question in pathology, which was this: May not, by some functional disorder, the physiological storage of bile be converted into a pathological stasis and thus become the cause of a more or less serious ailment of some part of the biliary system? I am inclined to answer this question in the affirmative. We are confronted with many possibilities to which I see no objection, derived either from theoretical considerations or from practical observations. The mechanism of bile storage and of bile discharge is depending on such a fine nervous adjustment that we can readily conceive a disturbance of it without the presence

of a concomitant palpable anatomical or chemical disorganization which played and still plays an all dominating pathogenetic role in pathology and medicine. We need, for instance, only to assume that by some mental excitement the tonic contraction of the sphincter of the common duct, at the period assigned for the discharge, does not become relaxed while the gall-bladder contracts within the usual normal limits; this would result in abnormal stasis within the biliary ducts which may lead to icterus—*emotional icterus*. Or both the muscle fibers of the gall-bladder and of the sphincter are abnormally strongly contracted, which would lead to biliary colic with consecutive jaundice without the presence of catarrhal conditions or of calculi, at least not in the first few attacks of comparatively short duration. When such disturbances occur often and are of longer duration these attacks of primary pathological biliary stasis may sooner or later lead to inflammatory processes and their consequences or to the formation of biliary calculi. Such conditions may set in sooner and readier when the individual passes through such infectious diseases of which we know that during that period the circulatory blood as well as the bile contain the causative organisms. Besides psychical influences (and perhaps disorganized internal secretions which are conducive to a disturbance of the emotional equilibrium) there are the influences of the partaking of food and of the character of the food which is partaken which may prove sooner or later to be pathogenetic factors in the formation of biliary diseases. Continuous fasting for many days, or even weeks, may be of lesser significance, since the consecutive stasis may be corrected by a gradual absorption of the stagnant bile and a great reduction or complete cessation of secretion of new bile. But simple infrequent feedings, say the partaking of only one meal or even two meals a day, may very gradually finally lead to pathological consequences. The accumulation of the bile in the gall-bladder and in the ducts for longer periods, and the continued repetition of these periods may gradually reduce the normal resisting power of the corresponding mucous membranes which may become a serious pathogenetic factor, in case the individual has passed or is passing through an infectious disease. Furthermore, taking the above-mentioned experiments of Rost in consideration, the abstaining from taking proteins or partaking them infrequently or in minimum quantities may prove to be another factor in the pathogenesis of biliary diseases.[1]

[1] About a decade ago feeding experiments were conducted by Chittenden on a squad of men to prove the fact that the high dietary calories, as required by Voit and his followers, were unjustifiable and that men and animals may show a normal metabolism and be apparently in perfect health even when partaking of only small amounts of proteins. In my paper on the "Factors of Safety" I objected to the conclusion that the minimum amount of food which shows no immediate palpable evil results is the ideal diet; the minimum should not be identified with the optimum. It is perhaps worth while to record the fact *that one member of the above-mentioned squad*

As stated above, we do not know yet sufficiently about the physiology of bile storage and bile discharge. Bile secretion and the actual physiological effect of bile I have not taken at all into consideration in the present discussion. When we shall know more of the physiology of the mechanism we shall know a good deal more about the pathology of biliary diseases. I belong to those who believe, and often said it in print, that the knowledge of physiology and an understanding of its disorders is of special importance to clinical medicine. Pathological anatomical changes are often only the result of a disease and are of a static nature and unchangeable. Physiological changes are of a dynamic nature, are often reversible, and clinical medicine has well-founded reason for the hope that by perseverent, intelligent research we shall gradually find more and more remedies and ways by means of which we shall be able to reverse various diseases—to cure them. In this paper I attempted to show that there is a general physiological principle which I termed the law of contrary innervation, which plays a leading part in the mechanism of storage and discharge of bile and that a disturbance of the fine adjustment may be a pathogenetic factor in various biliary orders. I have also shown further that very infrequent feedings and especially the avoidance of food which is capable of sending to the duodenum peptones and albumosis may lead to a pathological biliary stasis.

From these considerations it follows that even in health it is advisable to partake comparatively frequent meals, although they need not consist of large quantities, and that the meal shall contain foods which readily send peptone and albumosis into the duodenum. This principle ought to be especially observed in infectious diseases during which the bile contains the causative organisms. In typhoid, for instance, food should be administered every two hours and the food should contain materials which may readily be converted into peptone and albumosis. The frequent elimination

who followed out the minimum diet more conscientiously than any other member, and for a longer period, had the misfortune to pass through an attack of typhoid fever. For the last few years he complained of pain in the region of the liver, and recently was operated on for gall-stones. Another faithful member of that squad has been complaining for several years of indigestion and of pains in the right upper quadrant. I am not familiar with the details. I understand the trouble was diagnosed as duodenal ulcer. We are reminded of the dictum of William Mayo: that when there is trouble in the right upper quadrant only a laparotomy will reveal the exact cause of the trouble. I have not followed out the fate of the other men of the experimental squad who, by the way, did not seem to be overzealous in the performance of the experimental diet, nor did they follow it out longer than they had to. It is apparently insufficient to draw final conclusions from the *immediate* effects of a definite experimental diet; the fate of such individuals ought to be followed out for many years. What effect the "meatless days" and other dietary instructions may have in the future upon the races of belligerent Europe cannot be learned at present from the proficiency in school work and other immediate effects. The future might provide us with unexpected lessons, if we are alert enough and willing enough to connect events occurring so far apart.

of the bacteria-laden bile may be the means of preventing the development of cholangeitis and cholelithiasis. The direct administration of peptone in capsules may be useful in infectious disease as well as in some of the biliary disorders.[2]

THE DIAGNOSIS OF GALL-BLADDER DISEASE.[1]

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During the four years 1912, 1913, 1914, and 1915, detailed records were made of 1708 patients who presented themselves for diagnosis and advice. All of these came because they had some disorder of internal organs, most of them of the digestive organs. Among these 1708 cases there were 56 in which a diagnosis was made of disease of the gall-bladder; and a study of these shows not only the forms of gall-bladder disease commonly met with, but the variations of each form likely to occur.

I. Chronic Cholecystitis. Probably chronic cholecystitis always precedes cholelithiasis. Gall-stones form, according to present day opinion, because infection of the gall-bladder mucous membrane, increased secretion of mucus, desquamation of epithelial cells, and excessive production of cholesterin make their formation possible. There may still be room for discussion as to whether microörganisms enter the gall-bladder from the intestine, along the bile ducts; or from the blood stream, excreted into the bile from the liver. There may be reason for doubt as to whether colon bacilli, typhoid bacilli, streptococci, or staphylococci most often cause the infection. But there seems no reason whatever for skepticism as to the fact that low-grade infection of the gall-bladder and chronic inflammation so

[2] According to the view taken in this paper some cases of jaundice and of biliary colic have their origin in the fact that the sphincter of the common duct is abnormally contracted and does not become relaxed, as it physiologically should, during the contraction of the gall-bladder. In experiments with magnesium sulphate I observed that the local application of a 25 per cent. solution of that salt upon the mucosa causes a completely local relaxation of the intestinal wall. It does not exert such an effect when the salt is administered by the mouth, that is, when it has to pass through the stomach before it reaches the intestines. The duodenal tube, however, apparently has reached an efficient practical stage. I make, therefore, the suggestion to test in jaundice and biliary colic the local application of a 25 per cent. solution of $MgSo_4$ by means of the duodenal tube. It may relax the sphincter of the common duct and permit the ejection of bile and perhaps even the removal of a calculus of moderate size wedged in the duct in front of the papilla of Vater. Twenty-five cubic centimeters of the solution as a dose for an adult will bring no harm. For babies the dose should not exceed 4 c.c. The procedure could be developed into a practical useful method.

[1] Read before the Stanislaus County Medical Society, at Turlock, Cal., September 8, 1916.

induced underlie the formation of gall-stones. It follows, therefore, that a variable period must elapse of weeks, months, or even years during which chronic cholecystitis exists but cholelithiasis does not. In fact, no positive clinical evidence is ever furnished that gall-stones are present until they attempt to escape, giving rise to the dramatic set of events known as biliary colic. Before the occurrence of an attack of such characteristic pain, no other diagnosis can be made than that of chronic cholecystitis; and even after such attacks no gall-stones may really be found at operation because the same biliary colic may be caused by acute exacerbations of the chronic inflammation, with blocking of cystic and common duct by inspissated bile and by spasm. The symptoms that precede such attacks of pain, whether the pain be caused by stone or not, are the symptoms of chronic cholecystitis, and should be recognized before biliary colic calls attention unmistakably to the seat of disease. What then are the facts in the history and physical examination of any case that justify the conclusion that chronic cholecystitis exists? In the series of cases observed, this condition was diagnosed 17 times. In 8 of these operation was done and in 7 the diagnosis was verified; while in the remaining case that came to operation no disease of the gall-bladder was found, but a chronic appendicitis. In the other 9 cases diagnosed there seemed as much reason for the conclusion as in the seven that were verified by operation; and a review of the 17 will therefore show what the basis is for such a diagnosis.

The symptoms of chronic cholecystitis are of two sorts: (1) *local*, pointing direct to gall-bladder; (2) *reflex* disturbances of digestion, suggesting the stomach as the seat of disease. Sometimes one group of symptoms predominates, sometimes the other; most frequently there is a mixture of both. The following case history calls attention particularly to the local manifestations. The patient, a young woman, aged twenty-six years, when seen first in January, 1912, complained that for six years previous she had had numerous attacks of pain in her right side, varying in severity, brought on by over-exertion, or by jarring, such as riding in an automobile. Between attacks she complained of a sense of weakness in her side; of a pulling down feeling there; of pain in her side on reaching up with her right arm; of something crowding her just at the right costal margin; and of inability to sleep with comfort when lying on her right side. Finally, in December, 1911, her pain grew worse than ever before, fever developed and for the first time she became very yellow all over her body, the fever and jaundice lasting for several days. She had a good appetite and no food distress between attacks, but a constant sense of beating and throbbing in her side and the whole side felt bruised and tender after the December attack, clear to her armpit. The only definite physical finding was great tenderness and rigidity along the right costal margin, most noticeable about half-way between the nipple line and the right sternal border. With

medical treatment this patient's discomfort grew somewhat less, she gained a little in weight and strength, but her side continued to feel weak and swollen and tender and "thumped" on any exertion. Finally in August, 1912, she had another attack of severe pain in her right side, with intense nausea, with fever for four or five days, and jaundice that persisted this time for weeks. Operation was advised for chronic cholecystitis, with recurring acute exacerbations but was not permitted until March 1913. At operation there was found great thickening of the gall-bladder walls, and the contents were as black and thick as tar, but no stones were present. Drainage of the gall-bladder resulted in relief of all symptoms.

The following clinical picture better presents the mixed manifestations ordinarily found. A young woman, aged twenty-four years, seen first in February, 1912, had been ailing for six years with recurring attacks of pain in her right side; had been operated upon for chronic appendicitis and her appendix removed, in Philadelphia, in 1909, but without permanent relief; then again in May, 1910, in San Francisco, after constant nausea and pain in her side high up, at the costal margin, and after jaundice for two months, she was operated upon for drainage of her gall-bladder, when chronic inflammation was found but no stones. She remained well after that until the following December, when she began to have constant nausea and frequent vomiting; distress soon after food and a feeling of great fulness; dull aching pain localized on the right side up underneath the ribs, radiating under the right shoulder-blade, increased by exercise or any jarring such as horseback-riding; and inability to raise the right arm to pull a window down or to reach for anything on a shelf, without increasing her pain in the right side at the lower border of the ribs. Physical examination showed great tenderness at the spot where she felt her pain and very decided rigidity there; while stomach analysis proved that her digestive symptoms were due to a hyperchlorhydria. Further operation was declined; and while her condition was improved by diet and drugs, it was never restored to normal.

The local evidences of gall-bladder disease may be so pronounced as to speak for more than cholecystitis. As showing this resemblance of chronic cholecystitis at times to cholelithiasis and the difficulty in deciding whether the latter condition has really developed as a sequel, the following history is characteristic. A man, aged fifty-six years, seen in August, 1912, began five years before to have recurring attacks of pain and soreness in his upper abdomen, at first coming on months or weeks apart, but recently almost constantly, with only a few days' interval between; and at times these attacks had been severe enough to require an opiate for relief. Jaundice had come for a year past after every attack of pain, but usually lasting for only a few days. His appetite was very poor, all food caused distress, and he had lost 30 pounds in weight in the last year.

This man's physical examination was absolutely negative except for the evidences of moderate icterus in eyes and skin, with no tenderness or palpable mass in the gall-bladder area. Nevertheless, at operation his gall-bladder was the size of a goose-egg, attached to surrounding structures by many adhesions, its walls very thick, and its contents consisted of about two ounces of dark, tarry, viscid bile. No stones were found, only a few particles of coarse sand.

Even more puzzling is the following history, in its resemblance to that of gall-stones: A woman, aged forty-five years, seen in February, 1914, complained of pains in the right side of her abdomen, at the lower border of the ribs; these had annoyed her for years past, but worse for the past two years, coming at least once in six months, and for the past four months much more frequently, as often as twice a week. The pain came suddenly, most often at night, at the border of the ribs on the right side and radiating thence to the back under the right shoulder-blade; was very severe and cramp-like in character, only relieved by opiates, leaving her sore and tender, for a day or two afterward. No fever accompanied and no jaundice followed. The patient was obese, but no physical abnormality was found except extreme tenderness and rigidity at the right costal margin, over the site of the pain. Operation showed a contracted gall-bladder, containing thick, tarry material, with several fine dark crystals, but no stones.

Many of the cases of chronic cholecystitis come for advice for "stomach trouble," the reflex symptoms of digestive disturbance causing far more annoyance than the local gall-bladder manifestations, which are often overlooked entirely until questioning brings them out. The following history is typical of this group. A woman, aged forty-seven years, seen first in February, 1914, said she had suffered for years from what she called "intestinal indigestion" until her appendix was removed in 1905, after which this trouble ceased. But for about five years past she had had trouble with her stomach, varying in degree but never gone. Her complaints were that all food caused distress, persisting at times for hours; much belching of gas; frequent nausea for weeks or months at a time; with dizziness and vertigo and weakness. Inquiry revealed the fact that her worst pain was felt in her right side, at the margin of the ribs, running around to the back and under her right shoulder blade. Many days at a time she had this pain, with throbbing and a sense of too great fulness. Once, in association with this constant pain, she became jaundiced for a week. Examination showed rigidity and tenderness at the right costal margin, with extreme pain when the Murphy maneuver was attempted. A test-meal demonstrated the existence of a decided hyperchlorhydria (total acidity, 72; free HCl, 32; combined HCl, 20). This patient, like many others, was so obsessed by her stomach symptoms that she could not accept at once the diagnosis of chronic cholecystitis, and insisted on medical treatment;

until finally, in May, 1914, she had a definite acute exacerbation, with chill, followed by fever, and intense pain and constant throbbing in her right side at the costal margin. This led to operation which revealed many adhesions about the gall-bladder, its walls thick and tough, its lining membrane granular and thickened and its contents very dark and muddy. No stones were found. The stomach was normal in every respect. This patient slowly regained her health following drainage, her dyspepsia disappeared, she regained her lost weight and is today in perfect health.

One other case, seen in July, 1915, also well exemplifies the gastric side of the cholecystitis picture. This woman, aged twenty-nine years, complained most of her stomach—of a ravenous appetite, but that all food caused gas and belching, with distress and pain, relieved by getting rid of gas; these symptoms going on for years past, but getting more severe all the time; with constant soreness over the stomach and progressive loss of weight. When questioned about them, this patient recalled recurring attacks of severe pain in the pit of the stomach, preceded by much gas and bloating, when she would have to remove her clothing for relief and put on hot applications; the pain was not colicky, and never lasted over an hour. These attacks went back as far as eight or nine years; at first they came only once in three or four months; but for the past two years they had gradually become more frequent, and for the past few months had come as often as every week or every few days. Also, for the past six months, she had had much aching pain, and at times soreness at the margin of the ribs on the right side, always worse after a ride in an automobile. This was constant but variable in intensity. Examination showed a very tender rounded mass in the region of the gall-bladder, descending on deep inspiration from beneath the ribs. A test-meal showed only a moderate grade of hyperchlorhydria. Operation the following November showed extensive adhesions of gall-bladder to colon and liver, burying it and binding it down; it contained very dark, thick bile, but no stones. The gall-bladder was removed.

All of the 17 cases diagnosed in this series as chronic cholecystitis, like those that came to operation, showed this combination of pain and discomfort in the gall-bladder region, with more or less disturbance of digestion. Errors in recognizing the real state of affairs arise on the one hand from considering only the stomach manifestations, or, on the other hand, from misinterpreting the gall-bladder manifestations and calling all cases cholelithiasis. One other possible error lies in attributing both the local and the reflex symptoms to appendix disease; but this, in the writer's experience, is less probable than the opposite error of diagnosing cholecystitis when the real pathology is appendicitis. As regards the patients themselves, it is remarkable how often their minds are fixed on their disturbance of digestion to the exclusion of all else; and unless the diagnostician

is alert, he will get no complete story of the condition. Examination of the stomach most often reveals hyperchlorhydria as a cause of the dyspepsia; but sometimes subacidity and sometimes complete achylia. But no evidence usually exists of organic gastric disease, unless as a complication, while the history and the physical signs point direct to the gall-bladder as the seat of the real disease.

II. CHOLELITHIASIS. Stones may form in the gall-bladder and give no sign of their presence; the only symptoms being those already produced by the preceding cholecystitis. Such are the "inaugural symptoms" described by Moynihan as due to gall-stones, which frequently exist, even when ultimate operation shows no stones present but only a chronic cholecystitis. The clinical picture described as "biliary colic" is introduced into the history when a stone attempts to escape from the gall-bladder. Coincident with this event, either causing it or produced by it, an acute exacerbation is set up of the chronic inflammatory state; and it is difficult to say in any given attack how much of the symptoms are due to one or to the other. But the suffering caused by one of these attacks is usually so violent and excruciating, so far in excess of any previous effects produced by the gall-bladder disease, that now if never before the seat of the trouble is recognized and no longer doubt as to diagnosis can exist. In the series of cases here reported, cholelithiasis has been diagnosed 37 times; and in 20 of these operation has been done. In 17 by operation the diagnosis was proved correct; but in 3 no disease of the gall-bladder was found, while the real pathology was a chronic appendicitis. The cases of cholelithiasis with long intervals between attacks of biliary colic are loathe to accept operation, particularly if the intervals are characterized by fairly good health and freedom from digestive discomforts; hence the fact that out of 37 cases diagnosed, only 20 have come to operation. Many problems are presented, after all, in the recognition of cholelithiasis, because many variations occur in the clinical history, in the findings on physical examination, in the coincident gastric conditions and in the details of recurring attacks of biliary colic, even in the same individual. An account of some of the cases observed will best bring out the facts upon which we depend for diagnosis, and will show that there is no exact typical always recurring clinical picture.

A man, aged fifty-five years, seen in October, 1912, had his first attack of severe pain in the upper abdomen eight years before; and since then only three similar ones, lasting in each instance only a few hours at a time. In the interval between these he had occasional periods of indigestion, but much of the time no trouble at all. The site of his painful attacks was the pit of the stomach; the pain radiated through to the back, under both shoulder-blades, never around either side; was very intense, colicky and spasmodic, so that he felt at times as if he could not stand it any longer; it lasted a variable time, depending on his ability to get the opiate he required;

he never vomited during the attack and was never nauseated, but always perspired freely; never had been jaundiced after any attack, but was always left sore for several days in the pit of the stomach, not over his liver or right costal margin. His stomach trouble consisted of heaviness and fulness almost immediately after eating, with at times a dull pain; but no belching of gas, no heartburn or water-brash or nausea or vomiting. This man's stomach showed a decided hyperchlorhydria but no other abnormality; and his abdomen was slightly tender and tense at the right costal margin, but no palpable mass could be found there or elsewhere. For a long time he refused operation, convinced that his stomach was the seat of disease. Later on some of his attacks of pain were accompanied by chill and fever and the passage of very dark urine; the painful attacks grew more frequent in spite of medical treatment; he lost continually in weight and his indigestion became constant. Finally, in March, 1913, he submitted to operation. Many adhesions were found about the gall-bladder area, of the liver to the abdominal wall, of the gall-bladder to the duodenum and to the stomach; with one particularly strong adhesion between gall-bladder and duodenum, just beyond the pylorus, as if at sometime a stone might have perforated and escaped that way. The gall-bladder itself was very white, tough, and thick walled, and contained probably fifteen stones of varying sizes and shapes, old, hard, angular and with many facets.

The foregoing history shows how biliary colic may deviate from the orthodox text-book description. The pain was always epigastric, never radiated along the right costal margin, and was felt with equal intensity all over the back, beneath both shoulder-blades. Jaundice never accompanied or followed, neither was there vomiting. The man's own conviction, from his repeated spells of indigestion and the site of his painful attacks, was that his stomach was the primary seat of disease. Such a history might easily deceive, particularly in the absence of definite physical findings in the gall-bladder area and the presence of a persistent high-grade hyperchlorhydria.

Another common source of error is the impression that cholelithiasis occurs only in middle age or later. The following case demonstrates the incorrectness of such a belief: A woman, aged twenty-six years, seen in August, 1912, complained of stomach trouble. Beginning at the age of twelve years, she had repeated attacks of abdominal pain diagnosed appendicitis, recurring once or twice a year, until finally she was operated upon in 1908 and a purulent appendix removed. For a time after that she seemed well, but within a few months her attacks of pain in the upper abdomen began again, and she had had more of them during the past year than ever before. The attacks began with pain in the pit of the stomach, radiating usually up into the chest and not to the right side; this pain at times was very intense, and on some occasions required opiates; was colicky in character; came on frequently at night,

arousing from sleep; lasted for several hours unless an opiate was given; no nausea or vomiting accompanied and no jaundice had ever followed; there was some tenderness over the epigastrium at the time, but not afterward. As regards digestion, she complained much of heartburn, waterbrash, and sour stomach, but not of belching or nausea or pain. No pain was caused in the right side by jarring. This patient was well nourished, of good color, and no abnormality was found anywhere on physical examination, not even in the abdomen. The costal grooves showed no difference in tension, and there was no fulness or tenderness over the gall-bladder area. The stomach analysis was within normal limits and the stomach normal in size. Roentgen-ray plates revealed no abnormality. The history in this case was diagnostic, but there were no confirming signs. The patient, however, continued to have her recurring attacks of pain until operated upon in October, 1912, when the gall-bladder was found firmly adherent to the transverse colon, the pylorus, the duodenum, and in fact to all adjacent tissues; and it contained approximately 250 stones.

One grows accustomed after a time to the variations that occur in the story of biliary colic and can almost make the diagnosis of gall-stones by a careful history alone. There certainly is no more important element in the diagnosis than a painstaking, detailed account of the patient's attacks. Physical examination is often surprisingly negative in comparison with the pathology ultimately found; because the gall-bladder is tucked up beneath the ribs, where it is inaccessible to palpation. The history, therefore, becomes of great importance in any case, but as no two cases are exactly alike, there is, as before stated, no constant, unchanging clinical picture. The attack frequently begins at night, but it may begin at any time of day; the pain is usually located in the right hypochondrium, but may be most intense in the epigastrium or the left hypochondrium; usually it radiates along the right costal margin, to the back, under the right shoulder-blade, but may radiate instead directly through between the shoulder-blades, upward into the chest or downward into the abdomen; jaundice is not at all a regular accompaniment, and its absence is no bar to diagnosis; even when it occurs, it may be slight, transitory, and overlooked, while reflex symptoms, such as vomiting, may never occur at all. Fever may or may not accompany the attack of pain. No age is exempt from gall-stones, though they are more common in middle life; and they may occur in lean individuals, though more often in the obese.

The following is comparatively a typical history: A woman, aged fifty-one years, seen in July, 1912, began about five years before to have attacks of pain in the upper abdomen. These came once a month or even as often as once a week, but she might go two or three months without one. In the interval between attacks she felt perfectly well. Gradually her attacks had become much more

frequent; sometimes occurring every day or even twice a day. The attack came very suddenly and ended very suddenly; the pain was felt in the epigastrium and across the upper abdomen; its radiation was to the back and under the right shoulder-blade; its character was colicky and griping; its intensity was very great at times, but all attacks were not equally so; its duration was variable, usually about an hour or two. Attacks appeared to bear no relation to food; they might precede or follow a meal. During the attack she was nauseated but rarely vomited. With one attack, about two years before, distinct jaundice followed; and during one attack, while under observation in hospital at the time she came for diagnosis, her eyes showed decided yellowness of the conjunctivæ, the skin over the entire body became slightly yellow, her urine a dark brownish black, and her feces very light yellow. This patient was fat, of good color, and appeared well. No rigidity or tenderness could be elicited over the gall-bladder area. Her stomach showed no abnormality in size, position, or secretion. In fact, no evidence existed, except in the clinical history, that any serious pathological change had occurred in this patient's body. Yet at operation her gall-bladder was found small, shriveled, and shrunken, and contained two large stones, upon which it had contracted so that they completely filled it. Gall-bladder and stones were removed, and her pains never recurred.

So many of the cases of gall-stones present the same story over and over, with only minor variations, that it becomes monotonous to review their histories. It is interesting, however, to note the unusual variations from the ordinary, because these help us to recognize similar atypical cases another time; such is the following: A woman, aged forty-eight years, when seen first in June, 1913, had had "stomach trouble" for fifteen years past, worse for the past ten years. She had this trouble all the time, but also every month or two very violent attacks of pain, so severe as to require morphin. These started suddenly, and she felt as if in a vise, from the pain in front and back. At the outset she felt distended and full of gas. Then came the violent pain, steady, continuous; awful pressure with no intermittence, persisting until morphin was given. Attacks always began soon after a meal, and always seemed due to some food eaten, particularly to sour foods, as fruits and pickles. Between these attacks nearly all food distressed. Appetite was good, but she feared to eat. Distress came on an hour or two after eating; she filled up with gas and seemed much distended; and there was much belching. Such distress persisted for hours after food was taken. In the effort to get relief from these troubles she had been operated upon in September, 1912, for repair of lacerated perineum, suspension of a retroverted uterus, removal of both ovaries and tubes, release of intestinal adhesions, and removal of appendix. After this operation she was better for a time, but not for long. Therefore, in

February, 1913, another operation was done, for relaxation of vaginal walls and prolapsed uterus. But the following April she had another of her severe attacks of pain, and since then her stomach trouble had persisted constantly. This patient was obese and sallow. Her abdomen showed the characteristic rigidity and tenderness in the right hypochondrium, and her stomach a moderate hyperchlorhydria. In view of the history and findings a diagnosis was made of cholelithiasis, biliary colic, and reflex hyperchlorhydria.

Now comes the history that makes this case remarkable. Under routine medical treatment her attacks of pain disappeared and her stomach trouble ceased entirely for nearly three years. But, nevertheless, during these years she was never well. At one time she had a severe siege of "muscular rheumatism." She remained continually tired, exhausted, and anemic. In January, 1915, she had another attack of severe muscular pains in her chest wall. Later on she complained of constant aching and stiffness in her back and down both limbs, so severe as to prevent sleep. She had much headache, lost weight and strength, and in general felt wretched. Finally, after persistent suffering with pain in the back, headaches, and aching in the limbs, in April, 1916, her stomach trouble recurred and then she had a violent attack of her old pain. This led to immediate operation, and to the removal of one large gall-stone over three inches in circumference, another as large as a large acorn and about that shape, three others averaging an inch in circumference, and about 100 others, pea size or smaller, with very black, thick, infected bile. It seems clear that all the conditions arising during the three years' quiescence of the biliary colic were due to sepsis taking its origin from the gall-bladder, for all have entirely disappeared since the operation.

Finally is selected for review a case that calls attention to a complication of cholelithiasis, always present as a possibility, with every attack of biliary colic. A man, aged sixty-two years, seen in January, 1914, had been awakened at 2 A.M., on January 9, with intense pain in the pit of his stomach, radiating to his right side and through to his back. This lasted all day, like colic, and very severe; persisting also during the tenth day, with intermissions, but absent during the eleventh and twelfth days, only intense soreness remaining at the site of former pain. Early in the morning of January 13 he had another very severe attack, sudden in onset, at the right side of the abdomen, just at the edge of the ribs. On the afternoon of the ninth day he had a very severe chill, followed by fever and sweat; another such paroxysm at noon on the tenth day; a third on the eleventh day; a fourth during the forenoon on the twelfth day, with temperature rising to 105°; and a fifth late the same evening. Between these paroxysms the temperature became normal, falling with the profuse sweat. The patient was a very large, obese man, with slight icterus in conjunctivæ and skin. The abdomen showed ful-

ness and tenderness in the right costal groove, with moderate distention of the whole abdomen. Immediate operation advised revealed an acutely inflamed gall-bladder, containing purulent secretion, and 151 gall-stones, one of which had perforated the wall near the cystic duct and set up a localized peritonitis.

No reference has been made in the course of these case reports to *roentgen-ray examination of the gall-bladder*, because it is only very recently that improved technic has made it possible to obtain results in demonstrating the presence of gall-stones. At this time it is claimed that in about 50 per cent. of all cases, gall-stones will cast a shadow, so that it is undoubtedly in order to submit every suspected patient hereafter to an expert radiographer for his investigation. The writer has done this during the past year, but from the experience so far acquired the conviction is very strong that a negative roentgen-ray plate cannot be taken to exclude gall-stones, nor can a positive report always be accepted as conclusive; for in one recent instance in which the roentgen-ray plate showed shadows interpreted to mean gall-stones, no stones were found at operation.

As regards *stool examination for gall-stones* it is useless except soon after an attack of biliary colic, and very often negative then. The time so spent is not well repaid, because absence of gall-stones from the stool does not prove their absence from the gall-bladder; and the discovery of a gall-stone in the feces does not tell whether others remain in the gall-bladder or not. In the writer's experience, time is much better spent in sifting the history of these patients than in sifting their stools.

In considering the conditions that may be confused with gall-bladder disease and lead to error in diagnosis, *chronic appendicitis* stands out conspicuously above all others. Among the chronic cholecystitis diagnoses that came to operation, one proved to be chronic appendicitis; and among the cholelithiasis cases, three shared this fate. A description of these will show how difficult it is never to err. A woman, aged forty-four years, seen in March, 1912, stated that in the preceding August she had an illness characterized by slight pain in her right side and soreness just at the lower border of her ribs. Her skin became yellow and remained so for several weeks afterward. She had no fever at the time so far as she knew, but lost her appetite, and food distressed her. The whole attack lasted about two weeks. She had never been well since, gradually losing in weight and strength, and with constant stomach trouble—poor appetite, a sense of pressure and fulness after food; but no actual pain, no nausea or vomiting, no belching or waterbrash. She complained also of a sore spot and of darting pains in her right side, at the lower border of the ribs, present off and on for five years, but much worse and more constant since August. She had been a little jaundiced ever since her acute attack, sometimes more, sometimes less, but never so much since as at the outset. Under observation in

hospital she complained of pain in her right side, which she described as throbbing in character, a pressure, sense of tightness and clutching, severe enough to keep her awake at night; and in locating these sensations she pointed to the right side, at the lower border of the ribs, where she said she had, for months previous, a constant sense of soreness. She was obese; sallow but not jaundiced; with no mass palpable in the abdomen, but decided rigidity in the epigastrium and right hypochondrium, and with extreme tenderness on deep palpation, particularly over the gall-bladder area. The stomach was normal in size and position, but showed a definite hyperchlorhydria. The urine showed no bile and the feces no lack of it. At the operation advised for chronic cholecystitis, no evidence of disease was found in or about the gall-bladder, but a chronic appendicitis, with concretions within the lumen.

A man, aged fifty-one years, seen in March, 1913, complained of illness since the preceding October, characterized by loss of appetite, much gas formation and belching, frequent nausea, retching and vomiting after eating, and much pain in the right side, at the border of the ribs. This pain was constant for weeks at a time, but at times went away entirely. It was severe in intensity, but never like colic; it had been felt also in the back, on the right side. At the outset in October he had a chill, followed by fever, with terrific pain, requiring morphin repeatedly; this pain was located in the pit of the stomach at first, but later in the right side like the subsequent attacks. With the first attack he had no jaundice, but with a subsequent one in January his skin did become yellow for weeks. His abdomen showed rigidity and increased tension below the right costal margin and great tenderness on Murphy's maneuver, but no abnormality over stomach or appendix. The test-meal gave a normal secretion. Operation was advised for cholelithiasis. When it was performed the subsequent October no disease of gall-bladder was found, but a suppurative appendix, with a large abscess behind the cecum.

A woman, aged thirty-eight years, seen in May, 1913, very large and obese, gave a history of recurring attacks of abdominal pain, the first one seven years before, then about twice a year until the previous September, since then about every two months or six weeks. The attacks came with no warning at all, lasting until morphin was given, but never over a few hours. Her description of these attacks seemed typical of gall-stone colic. The pain was sudden in onset, like colic in character, as severe in intensity as when her baby was born; its site at onset was under the border of the ribs on the right side or in the pit of the stomach, radiating across to the left under the ribs and ultimately clear around into the back, so that it made a complete circle. Usually vomiting accompanied the attack, sometimes at the onset, sometimes after an hour, with repeated violent retching and straining, lasting even after the pain was gone. Attacks might occur in the early morning, rousing from sleep, or before

breakfast, often during the forenoon, never at night. Sour food would bring on an attack, as salad, pickles, or logan berries. She had never noticed any yellowness of the skin after an attack, nor any change in urine or feces. In the interval between she felt perfectly well; had an enormous appetite, ate very heartily, and it took a great deal of food to satisfy her. Her bowels were always constipated and never moved without aid. Physical examination was entirely negative, as regards stomach, gall-bladder, and appendix, and the abdomen seemed normal in every way. Suspecting gastric crises, careful investigation was made of all reflexes but no abnormality found. Stomach contents showed only a slight increase in acidity. Feces showed no ova of parasites. The Wassermann reaction was negative. The diagnosis made was cholelithiasis, with recurring attacks of biliary colic. At operation no disease of gall-bladder was found and no stones; no disease of stomach or duodenum, but the appendix was slightly enlarged, thickened, and clubbed. After removal it showed swelling of its mucous membrane, and its lumen filled with mucus. Smears of the latter showed many lymphocytes, a few polymorphonuclear leukocytes, a moderate number of short Gram-negative bacilli (probably colon bacilli), no other bacteria; sections showed some hyperplasia of lymph follicles, fibrous thickening of submucosa, areas of cellular infiltration, and some newly formed fibrous tissue in the subserosa. The pathological diagnosis was, therefore, chronic appendicitis. Clinical proof of its correctness was given by the fact that after the operation all attacks of pain ceased.

The last case of this sort presented itself in October, 1915. The woman, aged thirty-seven years, told the old familiar story of attacks of abdominal pain, recurring at intervals for fifteen years past, and gradually growing worse. At first these came about once a year, but they grew more and more frequent, until now they come as often as once a month. The pain appeared suddenly, with a feeling of bloating and distress in the pit of the stomach, gradually becoming very severe, and radiating to the right side and into the back. It was colicky in character and so intense as to require morphin for relief. Sometimes it lasted twenty-four hours, sometimes only two or three. She was always left very sore afterward, over the site of pain, as if someone had beaten her. She had never grown yellow with any attack, nor had her urine become dark or her feces light in color. At times she had chills and fever at the time of her attack, but only for a short time and not with all. She always vomited with any severe spell. Between times she had no digestive trouble, but was always constipated. This patient gave no signs of rigidity or tenderness or palpable mass over either gall-bladder or appendix. Her stomach contents showed a subacidity. Advised operation for gall-stones, she finally submitted to it in April, 1916. No disease of gall-bladder was found, but a chronic appendicitis; the

appendix was much enlarged and swollen and its lumen distended by pus.

How to avoid the error made in these cases is not altogether clear. Both conditions give rise to recurring attacks of abdominal pain. Ordinarily we depend upon the site of pain and tenderness, the radiation of pain, its character and intensity, to distinguish one from the other. But as shown by these histories, the clinical picture is not always definite. Either may give rise to chill and fever with leukocytosis, to jaundice, and to vomiting as coincident manifestations; and while appendicitis may have pain referred to the right costal margin and back, gall-stone colic may exceptionally cause pain referred down into the right side of the abdomen. It seems inevitable, therefore, that occasionally one will be mistaken for the other, in spite of every care.

Other conditions that must be distinguished from biliary colic are gastric and duodenal ulcer, gastric crises of tabes and renal colic. As regards ulcer, the error is conceivable; but the characteristic history of chronic gastric ulcer, the findings by physical examination, stomach analysis, and roentgen-ray plates should serve at least to exclude cholelithiasis, even though other possibilities for error remain. Examination of the reflexes, and Wassermann reaction, with lumbar puncture in any dubious case, should remove all doubt as to gastric crises. Renal colic has such a very different history as regards urinary symptoms and signs, and investigation is now so definite in its results by cystoscopy, ureteral catheterization, pyelography, and roentgen-ray plates, that one can rarely fail to recognize the underlying kidney disease if the colic attacks are renal in origin.

III. Cancer of the Gall-bladder. Two cases of this were met with in the series, one discovered as a result of operation and the other diagnosed before operation. The first case was a woman, aged fifty-two years, who had the usual typical cholelithiasis history, of recurring attacks of pain in the upper abdomen, for ten years previous, absolutely characteristic of biliary colic, except that she had never had jaundice with any attack, nor fever. The right costal groove was more full than the left, more rigid and very tender, but there was no palpable mass. Stomach analysis showed a decided hyperchlorhydria. At operation, advised for gall-stones, there were found many adhesions around the gall-bladder and great thickening and hardness of its walls, suggesting malignancy; with about thirty stones of various sizes and shapes. The stones were removed and the gall-bladder excised. The pathologist reported later on that sections from the gall-bladder showed carcinoma.

Such a sequel of chronic irritation by gall-stones is not surprising, and it is remarkable that it does not more often occur. The clinical history and the findings by physical examination were all those of cholelithiasis and biliary colic, and no other clinical diagnosis could be made. The pathological examination of removed tissue was what

showed the real condition, not suspected previous to operation. However, in the other case of cancer of the gall-bladder met with in this series the diagnosis could be made clinically. The patient was a woman, aged sixty years, seen in May, 1914. She complained of a lump in her right side, discovered about four weeks before. Her past history gave no evidence of attacks of biliary colic or of any chronic indigestion. She began to suffer with pain in her back several months before, but the lump in her side was only recently found. There had been some pain in this region but no severe suffering. There had been no distress from food and no vomiting. She had grown very yellow, however, for two weeks previous, and had lost much weight. The patient was moderately jaundiced. In her abdomen was found a hard, smooth, tongue-like tumor, projecting from beneath the costal margin in the region of the gall-bladder, half-way to the navel. This was freely movable, not tender, not nodular, and could be grasped and moved from side to side. The liver was not increased in area; its lower border, palpable on deep breath, was not nodular or tender; and was found just beneath the costal margin, while the rounded tumor extended as low as the navel when the patient took a deep breath. On inflation of the stomach its greater curvature was found 10 cm. below the navel; no peristaltic wave was elicited or other evidence of pyloric obstruction; and the tumor was found to be distinctly to the right of the inflated stomach outline. Stomach contents showed a complete absence of free HCl and a total acidity of 6. The feces were clay color, putty-like in consistence, and showed an excess of fat. The urine was dark brown and showed bile. The Wassermann reaction was negative. Exploratory operation showed carcinoma of the gall-bladder and ducts, with metastatic nodules in the liver. The tumor felt was the enlarged gall-bladder, corroborating the impression given by clinical examination.

In conclusion, the most valuable lesson to be drawn from these case histories and from the experience acquired in obtaining them and the others in the group to which they belong is that the older methods of diagnosis are still the ones upon which we must rely. Painstaking efforts in eliciting an orderly and complete history not only of the patient's complaint but of all other incidents in the previous health; careful and intelligent use of the eyes, the finger tips, and the ears in acquiring information about the patient; and good judgment in sifting and interpreting the facts so obtained are still the methods that surpass all others in diagnosis. The newer laboratory and roentgen-ray aids are exceedingly valuable and never to be overlooked, but not to be relied upon as short-cuts and labor-saving devices to supersede the other means described, as seems to be too much the tendency in this day.

MEDICAL ASPECTS AND DIAGNOSIS OF DISEASES OF THE GALL-BLADDER.[1]

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The diseases of the gall-bladder in which we are most interested are cholecystitis and cholelithiasis. As an independent affection, inflammation of the gall-bladder is not common. In the majority of cases the gall-bladder is the seat of stones. Nevertheless, we must take cognizance of non-calculous inflammation. It is met with in typhoid fever, pneumonia, food-poisoning, etc., and manifests itself by tenderness in the gall-bladder area, by leukocytosis, and usually by jaundice. But even in apparently primary cases an unsuspected gall-stone may exist, as I once observed in a case of typhoid fever. As non-calculous cholecystitis, whether due to the typhoid bacillus or other causes, may closely resemble gall-stone colic, some physicians and surgeons, to avoid embarrassing explanations to the laity, no longer make a diagnosis of gall-stones, but merely of gall-bladder trouble. Surely it is an uncomfortable feeling not to find at operation the gall-stones one has predicted. In such an impasse one thinks of the practice of Conan Doyle's Dr. Winter, who carried stones in his pocket to meet these emergencies. Acute cholecystitis, especially the calculous form, is often of a fulminant character, producing all the signs of a severe septic infection with localized peritonitis. In one case, seen several years ago, in which the gall-bladder was filled with stones and pus, the onset was so acute as to suggest perforation of a gastric ulcer. In the diagnosis of such cases the history is of great moment; the patient generally has had attacks of pain in the upper abdomen and has suffered from indigestion and possibly jaundice. If the patient is a stout woman past thirty, the suspicion of gall-bladder infection is strengthened. On physical examination the gall-bladder in these cases can sometimes be felt, especially if a rolled-up blanket or pillow is placed under the back and the abdomen is palpated very lightly with the warmed hand. A large gall-bladder generally means a stone in the cystic duct. In such cases the liver often projects a considerable distance below the costal arch in the form of the so-called Riedel's lobe. Cholecystitis sometimes occurs in women of advanced years who never have had any gall-bladder trouble before. As I have seen it the attack has always followed a gross indiscretion in diet, and during the first day or two has resembled "ptomain poisoning." The symptoms are sudden epigastric pain with nausea and vomiting, prostration, and moderate fever. On the

[1] Read as part of a symposium at a meeting of the American Gastro-enterological Association, Washington, May 8, 1916.

second or perhaps the third day an enlargement in the liver region with great tenderness on pressure can be made out. This enlargement is evidently the swollen gall-bladder. A slight leukocytosis and congestion over the base of the lungs complete the clinical picture. Jaundice has not occurred in my cases.

I shall not dwell on the well-known symptoms of gall-stone colic except to say that the pain is much more often in the epigastrium than the text-books state. It may travel to the back, to the right side or to the left, and to either or neither shoulder. Nausea and vomiting while common are not rarely absent. A chill is of significance. Most patients complain of a sense of upward pressure during an attack of gall-stone colic. If a patient states that in one or more attacks the pain was so severe as to require a morphin injection, I take that as contributory evidence of biliary colic. Jaundice is not common; its presence is of great diagnostic value, its absence without significance. If every patient's urine is examined within forty-eight or seventy-two hours after an attack, traces of bile will not rarely be found. Many patients who declared that they never were jaundiced told me on inquiry that after an attack of pain they had passed very dark urine.

The pain in gall-stone colic may be left-sided. In a patient whom I saw several years ago with Dr. Farr the pain was never anywhere except in the left hypochondrium. Operation revealed a single large stone in the gall-bladder, with adhesions between the latter and the stomach.

An attack of pain in the upper abdomen which under other circumstances I would attribute to gall-stones, I should feel quite sure *was* due to that cause if the patient was a pregnant or puerperal woman. This is due to the fact that, as I have often observed, the first attack of biliary colic not rarely occurs during or soon after pregnancy.

It is always well in cases of upper abdominal pain of obscure origin to have the bowel movements searched for gall-stones. They are not infrequently found.

Pancreatic stones may cause symptoms similar to gall-stones. They are so rare that little need be said about them. It has been stated that the Roentgen ray shows pancreatic calculi more clearly than it does gall-stones.

Gall-stone disease may be simulated by floating kidney even to the point of jaundice. The discovery of a loose kidney and a history that the pain is relieved by lying down, and that the attack subsides with the passage of a large amount of pale urine would make us think that the attack was a so-called Dietl's crisis and not biliary colic.

In several instances I have been much perturbed by a difficulty in deciding between appendicitis and gall-stone. If the patient is young, thin, and spare, and has a history of some intestinal dis-

turbance, either diarrhea or constipation, at the time of the attacks, I should consider these facts suggestive of appendicitis rather than of gall-stones, even if the pain were near the costal border. Perhaps the sign pointed out by Dr. Aaron will prove helpful.

Surgeons are prone to draw very sharp distinctions between gall-bladder disease and gastric ulcer, no doubt because many of the cases they see group themselves readily under one or the other heading; but a physician seeing ambulatory cases in his office and in dispensary practice often has great difficulty in deciding whether a patient has gall-stone disease or gastric or duodenal ulcer. Many who complain of definite symptoms that seem to point to one or the other have neither, and are examples of so-called functional disturbance. I have seen several such patients in whom operation showed nothing to account for their symptoms.

The most difficult cases are undoubtedly those in which the gall-bladder is adherent to the stomach or the bowel, either as the result of primary disease of these parts—generally ulcer—or of repeated attacks of cholecystitis. We have, then, a fusion of hepatic and gastric symptoms, often with hyperchlorhydria, and it requires long and patient study to determine which is the underlying disease. A duodenal ulcer adherent to the fundus of the gall-bladder may produce symptoms scarcely distinguishable from gall-stone colic. In the diagnosis three things are necessary: A careful history, a roentgenologic study by an expert, and a physical examination, with all the modern laboratory investigations, including the use of the duodenal tube of Einhorn. Each of these methods may be counted upon to give about 30 per cent. of evidence. In cholelithiasis the Roentgen ray proves reliable, *i. e.*, shows the stone, in about 50 per cent. of the cases; it gives far better results in gastric and duodenal ulcer.

One of my patients, a physician, had symptoms of cholecystitis with tenderness in the gall-bladder area. The pains were so severe that he had taken to inhaling ethyl chlorid, and was using the drug in large quantities, $20 worth in a week. Operation showed a duodenal ulcer about to perforate, with the gall-bladder adherent to the base of the ulcer. In the patient's history the symptoms of duodenal ulcer seemed to be almost completely overshadowed by the secondary trouble in the gall-bladder.

One point of importance that has impressed me as of possible value in these cases is the persistence of the pain. Attacks of pain only of gall-bladder origin rarely last longer than a few hours or a day or two, while those of ulcer frequently last much longer. Gall-bladder attacks are more often nocturnal than those due to gastric ulcer, and are less related to the food intake. It is true, duodenal ulcer usually causes pain at night, but the pain is relieved by food, drink, or alkalies, and is much more regular in appearance, the patient being wakened from sleep almost at the same hour every

night. Many patients suffer from gall-stone colic whether they diet or not.

Sometimes cholangitis closely simulates gall-stone disease. In addition to pain there may be intermittent fever, with or without jaundice. Such cases are most perplexing. Sometimes there is nothing but an intermittent fever (Charcot's intermittent fever), which may extend over a period of weeks and even months. Jaundice may or may not be present. In one very protracted case that came under my observation, jaundice never occurred. Although gall-stone is the most natural diagnosis, the operation may reveal nothing but a gall-bladder filled with dark, thick, infected bile. Standing at the operating table one is inclined to think that the findings are insufficient to explain the clinical symptoms, and yet drainage of the biliary passages is all that is necessary to bring about a cure.

Gall-stone colic may be closely simulated by the crises of locomotor ataxia, the pains of which are often so severe as to require morphin injections. I believe it is a good rule in all cases of supposed gall-stone colic to test the knee-jerk, the station, and the pupils. As regards the last, it should be remembered that one might think an Argyle-Robertson pupil existed, when in reality the myosis was due to morphin. As a rule the gastric crises attacks are irregular in occurrence and extend over a long period (from several days to two weeks), and are not noticeably influenced by drugs, even by morphin. The Wassermann test is of value.

Pleurisy and pneumonia may counterfeit the picture of gall-bladder disease in a well nigh perfect manner. I recall the case of a patient who had been seized with pain in the right upper abdomen, jaundice following a day or two later. The attending physician had diagnosed gall-stone colic, but a careful examination disclosed lobar pneumonia.

Sometimes a sharp pain in the *back* or in the right chest *posteriorly* is a manifestation of gall-stone disease. I have seen several instances. The diagnosis is extremely difficult; one must be governed by the absence of renal symptoms, and perhaps by finding, notwithstanding that the pain is in the back, tenderness in the gall-bladder area.

Cases are common in which the symptoms are those of nervous dyspepsia. The patients never have severe pain, but suffer for years from "gas," belching, bloating, indigestion, and various neurasthenic manifestations. The fact that neither diet nor the rest cure to which many are subjected is of any permanent value should arouse the suspicion of organic disease, which might be ulcer, appendicitis, or gall-stones. To differentiate properly among these possibilities, especially between ulcer and gall-stone, is impossible by any rule of thumb. The clinician must in the last analysis be guided by his sixth sense, clinical intuition.

That there should at times be difficulty in distinguishing between

gall-stone disease and angina pectoris might seem incredible, yet it is true. Only a careful analysis of the history and a thorough physical examination will prevent mistakes. Apart from this indirect relation between gall-stones and angina pectoris, due to a remote similarity in symptoms, there are also more direct relations existing between the gall-bladder and the heart. The facts, as I have observed them, are about as follows: In a certain number of patients who have suffered from gall-bladder infection, often unknown to themselves, cardiac symptoms appear. These vary in character. Quite frequently a systolic mitral murmur is found, usually without any signs of cardiac failure. In other cases, in which the myocardium is evidently affected, the symptoms are those of sudden acute heart attacks characterized by dyspnea and cyanosis; in still others the attacks are as far as one can tell anginal in character. Now it is a curious fact that in nearly every case, at least in my experience, the heart symptoms have disappeared after operation. The importance of this phase of the subject is that one may consider the case one of a hopeless heart affection and overlook the causative factor, removal of which would cure the cardiac condition. On the other hand many a physician is deterred from advising operation on finding some cardiac disturbance or a heart murmur, because he looks upon it as a contra-indication to operation. In reality it is to be looked upon, unless too far advanced or clearly due to an independent disease, as a distinct indication for operation.

Lately attention has been called to a possible relation between infections of the gall-bladder and disease of the joints. Granted that multiple arthritis is in many instances a secondary process, there is no reason why the gall-bladder may not be the primary focus as well as the gums or the tonsils. In one case of arthritis deformans, in which a gall-bladder packed with stones was removed by Dr. Deaver, the joint condition was in nowise influenced. As the patient had had, however, the trouble for years, little was to be expected.

Much difficulty is presented by certain cases of chronic obstructive jaundice. While in middle-aged and elderly persons chronic jaundice is always suggestive of malignant disease, especially of the pancreas, it is possible to have such jaundice as the result of adhesions, stone, or chronic pancreatitis. I have sometimes thought that early itching was more common in malignant disease. If the gall-bladder is palpable the chances are against stone and in favor of external obstruction. Jaundice varying in intensity is nearly always due to stone in the common duct. There may not be in such cases any history of pain or at least any history of sharp colic. A patient with chronic jaundice may lose a great deal of flesh without having carcinoma. The significance of these facts is their bearing on treatment, a discussion of which is beyond the scope of this paper.

GALL-BLADDER DISEASE: A PROGRESSIVE INFLAMMATION: ITS TREATMENT, CHOLECYSTECTOMY.[1]

By Russell S. Fowler, M.D., F.A.C.S.,

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The title of this paper explains its purpose. The author believes: (1) that gall-bladder disease is a progressive inflammation from its incipiency; (2) that its treatment is early cholecystectomy before the disease has advanced beyond the gall-bladder. In furtherance of this purpose the subject has been treated in as simple a manner as possible, and it is hoped that with the aid of the illustrations its object will be accomplished.

The material for this study consists in upward of 100 gall-bladders presenting all degrees of pathological changes removed by the writer during the last ten months at the German Hospital and the Methodist Episcopal Hospital. For assitance in the miscroscopic study of the sections acknowledgement is extended to Doctors T. H. Dexter and W. W. Laing.

The inflammatory lesions of the gall-bladder so merge one into the other that it is impossible to secure specimens that are typical of one stage of the inflammatory process alone. So true is this, indeed, that it is quite logical to conclude that all are but different stages in a progressive inflammation. This progressive inflammation may be divided into three stages:

1. *Cholecystitis catarrhalis subacuta*, a first stage, where macroscopically the mucous membrane is but slightly affected and the rest of the wall not at all, but which presents microscopically characteristic changes. Grossly, the gall-bladder appears normal except for a more than usual velvety appearance of the mucous membrane. Stones may be present or absent. If present they are usually small. The aspirated bile is thicker than normal and more viscid. Microscopically (Figs. 1, 7, 8), the villi are broadened, elevated, and infiltrated with lymphocytes. This infiltration may extend into the wall of the gall-bladder. This stage I have described elsewhere as cholecystitis catarrhalis subacuta, with the added observation concerning the hypertrophied musculature, which latter seems to me to be of importance in placing this pathological picture in its true place as the earliest inflammatory stage in the progressive inflammation affecting the gall-bladder; for if the increased viscidity of the bile is due to increased activity of the mucous glands and partial though slight obstruction to drainage, then the hyper-

[1] Read before the Brooklyn Pathological Society, October 12, 1916.

trophy of the gall-bladder musculature naturally fits in the picture. Stronger efforts at expulsion are needed to empty the viscus, and until the musculature is invaded by the progressive inflammation, and finally impaired, the increased burden that is put upon it results in hypertrophy.

In addition, Luschka's spaces are somewhat dilated. In those cases in which no stones are found it is possible that they may have passed through the ducts. It is also just as probable that this is a pre-gall-stone stage.

FIG. 1.—Cholecystitis catarrhalis subacuta, showing a broadened villus elevated and infiltrated with lymphocytes. At the base the infiltration extends into the gall-bladder wall. The musculature shows hypertrophy. There is a somewhat dilated Luschka's space.

2. *Cholecystitis Catarrhalis Chronica.* Gross: Externally the gall-bladder does not present evidence of inflammatory change except for the gland at the cystic duct, which is usually enlarged. Stones may or may not be present. Here and there in the mucous membrane are minute yellow bile-stained areas of destroyed epithelium. These may be scanty or numerous. When plentiful they give a speckled appearance to the gall-bladder mucous membrane. Other areas show the characteristics of cholecystitis catarrhalis subacuta or appear normal, or the site of slight acute inflammation.

Microscopic (Figs. 2 and 3): The mucous membrane villi are thickened, the apices in places denuded of epithelium. It is these denuded areas which appear macroscopically as yellow specks.

This is the characteristic lesion. There is infiltration by lymphocytes of the mucous membrane and submucous tissues, and here

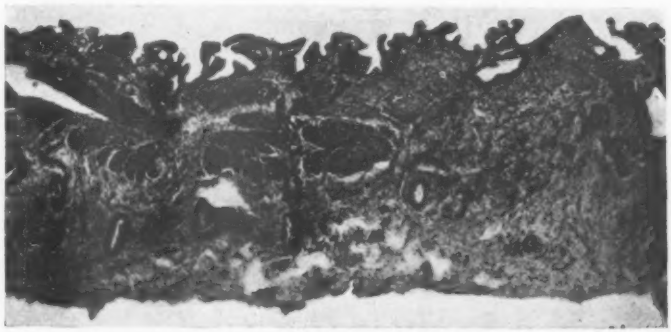

FIG. 2.—Cholecystitis catarrhalis chronica. The bases of the villi are broadened, the apices in places denuded. Lymphocytic infiltration of the mucous membrane and submucous tissues, and here and there of the hypertrophied musculature (beginning cholecystitis chronica). (Low power.)

FIG. 3.—Cholecystitis catarrhalis chronica, showing in detail the changes in Fig. 2. (High power.)

and there of the hypertrophied musculature (beginning cholecystitis chronica). Luschka's spaces are somewhat dilated.

Rarely is a specimen of cholecystitis catarrhalis chronica found which does not show characteristics in various parts of the specimen

FIG. 4.—Cholecystitis chronica. Lymphocytic infiltration into all parts of the gall-bladder wall. The musculature is markedly invaded. A villus stump is shown. (High power.)

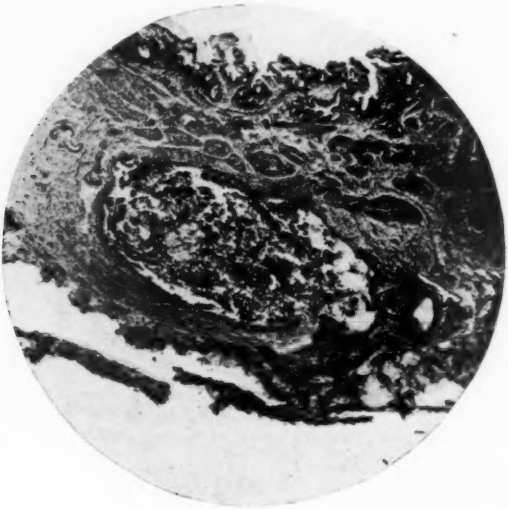

FIG. 5.—Cholecystitis chronica with stone formation in a Luschka space.

of the three stages of the progressive inflammation characteristic of gall-bladder disease—cholecystitis catarrhalis subacuta, cholecystitis catarrhalis chronica, and cholecystitis chronica. The distinction between the three is made on the predominance of the lesion appertaining to each. It is common, however, to find cholecystitis chronica without the first two stages.

FIG. 6.—Cholecystitis catarrhalis acuta. Though the papillæ including the tips are well covered with epithelium, the mucous membrane shows the following deviation from the normal: The cell outlines are for the most part obscured by cloudy swelling, though the nuclei are fairly distinct. There is a slight tendency to leukocytic infiltration in some areas, especially beneath the epithelium. A mild but distinct attempt at papillomatous formation is shown in the right half of the section. The muscle shows little if any change. Under the high power the infiltration beneath the epithelium is shown conclusively and there is also shown occasional and mild engorgement of some of the blood spaces in the papillæ, and the lymphoid areas are enlarged and engorged. The acute catarrhal differs from the subacute in the more marked changes in the mucous membrane which is hypertrophied and cloudy. The infiltration of the mucous membrane is intense.

3. *Cholecystitis Chronica.* Gross: Unmistakable evidence of chronic inflammation.

Microscopic (Figs. 4, 5, 9, 10): The entire gall-bladder wall is affected. The mucous membrane is lacking for the most part, being replaced by scar tissue; stumps of scar tissue represent what is left of the villi. There is infiltration of lymphocytes into all parts of the gall-bladder wall. The musculature is markedly invaded and more or less transformed into fibrous tissue. Here and there are areas of contraction where the process has been longest producing

macroscopically irregularities of the surface. It is logically what might be expected of a continuance of cholecystitis catarrhalis chronica. All stages of both the previous processes may be present, but mostly the picture is of chronic inflammation, with only an occasional area of mucous membrane.

The characteristics of this chronic inflammation are modified by pressure caused by chronic obstruction of the cystic duct. This occurs sufficiently often to be dignified by a separate subtitle, cholecystitis chronica cystica. Gross: such gall-bladders (hydrops) present unmistakable evidence of chronic inflammation, are

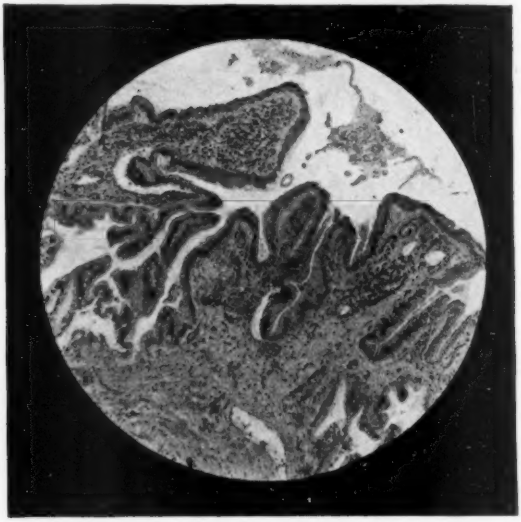

FIG. 7.—Cholecystitis acuta superimposed on cholecystitis catarrhalis subacuta. The bloodvessels of the muscular coat show an engorgement similar to that noted in the mucous membrane, and there is a distinct diffuse round-cell infiltration. (Low power.)

increased in size, sometimes enormously so, pale in color, the mucous membrane for the most part lacking, contents limpid fluid; wall thinned from pressure; obstruction usually by stone in the neck of the gall-bladder or in the cystic duct.

Microscopic: There is little if any mucous membrane present; what is found is flattened from pressure; the wall is formed of elastic connective tissue, with here and there muscle nuclei, and if the process is not too old, some muscle tissue.

These three stages—cholecystitis catarrhalis subacuta, cholecystitis catarrhalis chronica and cholecystitis chronica—form the large proportion of cases of gall-bladder disease. They are all

stages of the same inflammatory process, and are progressive. On them may be grafted other varieties of inflammation and carcinoma. Of the other varieties of inflammation we have:

Cholecystitis catarrhalis acuta (Fig. 6).
Cholecystitis acuta (Figs. 7 and 8).
Cholecystitis phlegmonosa acuta (Fig. 9).
Cholecystitis hemorrhagica acuta (Fig. 10).

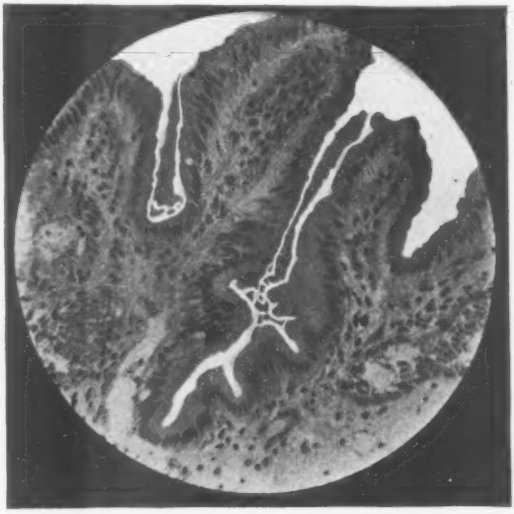

FIG. 8.—Cholecystitis acuta superimposed on cholecystitis catarrhalis subacuta. The villi are moderately but distinctly thickened. The bloodvessels of the mucous membrane are distended, and the villi show distinct leukocytic infiltration and some hemorrhagic extravasation. The epithelium of the villi including that of their tips shows distinct excessive proliferation. The lumen of the gall-bladder especially between the villi shows accumulation of mucus, leukocytes, red blood cells and detritus. (High power.)

Of these, the first may be present with but slight evidence of cholecystitis catarrhalis subacuta and no evidence of the other progressive inflammatory changes. Whether it precedes the subacute stage is, in my opinion, questionable. I believe that the sequence of events is: (1) cholecystitis catarrhalis subacuta (induced by interference with drainage and very mild infection); (2) cholecystitis catarrhalis chronica, the logical sequence of a continuation of the subacute stage; (3) cholecystitis chronica, the extension of the progressive inflammation already existent to other parts of the gall-bladder wall, together with a more pronounced inflammation and destruction of the parts previously involved in the subacute catarrhal and chronic catarrhal stages.

Of the remaining three—cholecystitis acuta, cholecystitis phlegmonosa acuta, and cholecystitis hemorrhagica acuta—none is ever present without evidence of cholecystitis catarrhalis chronica or acuta or cholecystitis chronica. An acute ulcerative process (cholecystitis acuta ulcerativa) may supervene on either.

FIG. 9.—Cholecystitis phlegmonosa acuta superimposed on cholecystitis chronica. This section shows an ulcer beginning in the mucosa and involving the gall-bladder wall. The mucous membrane is absent and at the area where it should exist there is a depression the surface of which is covered with leukocytes, red cells and detritus. The depression that represents the ulcer invades the muscular coat to a considerable depth. The muscular coat itself shows marked round-cell infiltration diminishing in direct proportion to the distance from the ulcerative surface. There is marked hyperplasia of connective tissue at the expense of the muscle bundles which are distinctly thinned out and separated by hyperplastic connective tissue. The entire gall-bladder wall is involved in an acute phlegmonous process superimposed on chronic cholecystitis, the existence of chronic cholecystitis being shown by the scanty muscle bundles with interposed connective tissue.

A chronic ulcerative process (cholecystitis chronica ulcerativa) may supervene from general pressure in hydrops or local pressure from stone in cholecystitis chronica.

Cholecystitis catarrhalis papillomatosa may be associated with cholecystitis catarrhalis acuta (Fig. 6) or cholecystitis catarrhalis chronica (Figs. 11 and 12). The author has found it once in both conditions. There is no reason why it should not be present in an otherwise normal gall-bladder or why a gall-bladder the seat of a papilloma should not also be subject to any other lesion.

Carcinoma is found engrafted on cholecystitis chronica (Fig. 13) and as cholecystitis papillomatosa malignum (MacCarthy) and cholecystitis catarrhalis carcinomatosa (MacCarthy).

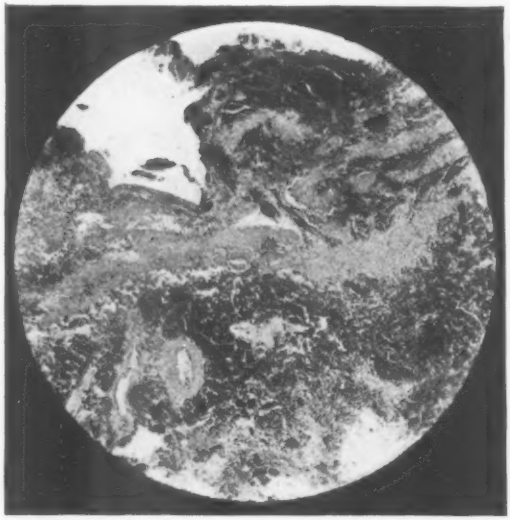

FIG. 10.—Cholecystitis hemorrhagica acuta superimposed on cholecystitis chronica. The mucous membrane is destroyed. The cells of the muscular coat show various stages of cloudy swelling and coagulation necrosis. Their protoplasm has a homogeneous appearance, their nuclei are indistinct and cell outlines are blurred or absent. Bloodvessels are distended with red cells and the tissue generally shows a marked extravasation of red cells and a diffuse leukocytic infiltration of the muscular wall, irregular in distribution, and with some tendency to localization in minute areas the prototype of microscopic abscess foci. If this section were considered by itself, the picture would be one of acute hemorrhagic cholecystitis, for the cloudy swelling of the muscle layers does not show any evidence of chronic inflammation, but section of other parts of the gall-bladder shows indubitable evidence of chronic cholecystitis. In some areas the mucous membrane shows coagulation necrosis of the single layer of columnar cells covering the papillæ. The cells appear granular, vacuolated and have lost definite outline, though the nuclei remain clear and sharply defined. In the papillæ and beneath these cells the papillæ show a marked hemorrhagic and leukocytic infiltration which involves also, to a less extent and in an irregular way, the muscular coat. Here also appears some connective-tissue hyperplasia indicating a chronic factor in the process. The tips of the papillæ are not denuded.

CONCLUSIONS. From a consideration of all of the above it is logical to assume that cholecystectomy is indicated in all diseases of the gall-bladder whether causing mild or severe symptoms. The operation is not done for the purpose of removing stones, for these may or may not be present, but for the purpose of removing tissue diseased as a result of a progressive inflammation. Just as soon as this inflammation has reached a point where the efficiency of the

gall-bladder is impaired and symptoms result, just so soon should the gall-bladder be removed.

Whether gall-stones are present or not is immaterial, for they are but one of the ways in which an already existent inflammation expresses itself. Personally, I do not believe there is any such thing as innocent gall-stones, but that they are the product of inflammation; that a gall-bladder once infected is always infected, and such a gall may become at any time a source of real danger to the patient through either acute exacerbation of the inflammation or through change to carcinoma during middle or later life.

FIG. 11.—Cholecystitis catarrhalis papillomatosa associated with cholecystitis catarrhalis chronica. (Gross specimen.)

Whenever possible, cholecystectomy should be performed early, not only for the reasons stated, but also because inflammations of the gall-bladder are, for the most part, probably always, recurrent and progressive, and if the inflammation is allowed to follow its natural trend at the very least there results adhesions, which in their turn produce annoying and in many instances disabling symptoms. So many operations have been performed on the terminal pathology of this condition, with an attendant high mortality and disability from already existing complications, that it is time for early operation to be advocated, because early operation at a time when the disease is limited to the gall-bladder means, at the very least, the

saving of much discomfort and inefficiency later in life. At all events the patient should be given an opportunity to choose in the early stage of the disease whether he wishes to continue to be subject to recurring attacks of so-called indigestion, with the added dangers of acute inflammation, and the final danger of carcinomatous change.

FIG. 12.—Cholecystitis catarrhalis papillomatosa associated with cholecystitis catarrhalis chronica. Note the slender base of the papilloma. (Low power.)

Formerly the high mortality of operations in this region was thought to be a bar to operative interference except in advanced cases; it is these very cases which are responsible for the high mortality; the disease should not be allowed to progress to this point. The lesion is initiated in the gall-bladder, and early removal of the gall-bladder will prevent the complications above stated,

and also will be attended with an extremely low mortality. This mortality should be no higher than that attendant upon operations for chronic appendicitis. Personally, I have never had a death from an operation upon the gall-bladder in which the disease was limited

FIG. 13.—Carcinoma superimposed on cholecystitis chronica. The portion of the gall-bladder from which this section is taken is very thick and dense. The section shows a great preponderance of dense fibrous tissue in which is a glandular or adenomatous type of carcinoma. There are also many areas of carcinoma cells infiltrating the fibrous tissue in a lawless way. These cells differ morphologically from those of simple hyperplasia in that the nuclei are large, irregular in shape and size, and have irregularly distributed chromatin. In contrast to this are found occasional normal glandular elements consisting of a tubular arrangement of a single layer of columnar or cuboidal cells, on a basement-membrane and presenting no deviation from the normal uniformity of size, shape and pigmentation of cells and nuclei.

to the gall-bladder itself. The cases in my experience which have succumbed have been those in which carcinoma was present or in which the disease had extended to the common and hepatic ducts, or in which there was gangreous inflammation and perforation.

PERICARDIOTOMY FOR SUPPURATIVE PERICARDITIS FOLLOWING PNEUMONIA.

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AND
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Operative intervention in wounds of the heart and pericardium has become so successful in recent years that it is reasonable to hope that surgery may be employed more frequently and more successfully in non-traumatic suppurative pericarditis. In order to stimulate discussion on this topic we present this report.

In a series of papers by American writers, Roberts,[1] 1897, Porter[2], 1900, Eliot,[3] 1909, and Rhodes,[4] 1915, there has appeared an exhaustive presentation of the subject of suppurative pericarditis, with a collection of the reported cases of pericardiotomy for suppurative pericarditis. "The cases numbered 86, of which 45 recovered and 41 died, a percentage of 52.3 recoveries against 47.7 deaths. In the complete series the infecting organism is reported in but 21, the pneumococcus was found in 9 cases, staphylococci in 4, streptococci in 2, the colon bacillus in 1, the Bacillus pyocyaneus in 1, and a 'double coccus' in 1." (Rhodes.)

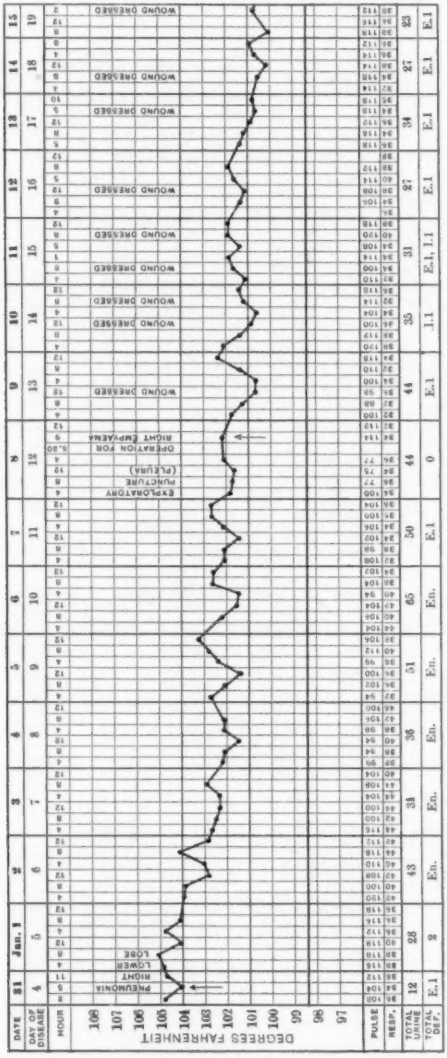
In connection with the case here presented, a case of pericardiotomy for suppurative pericarditis following pneumonia and empyema, we present synopses of the reports of similar cases. We also revised the detail of the technic of paracentesis pericardii and pericardiotomy.

Case Report.—Patient male; aged forty-seven years; occupation, engineer. Family and past history negative. Patient has been under the care of one of us (Camac) for various illnesses for the last fifteen years. These illnesses had no bearing upon the present attack. On December 24, 1915, at the office of one of us (Camac) patient was seen for a furuncle on the back of the neck, which was opened and dressed. On December 29, the wound was practically healed.

On December 30 the patient was seen at his house, and complained of a slight pain in the lower right axilla, on taking a deep breath. Examination showed a few very fine, almost inaudible crepitant rales at the right base, with somewhat harsh, vesicular breathing. Temperature, 104°. He was flushed and felt ill. Respirations,

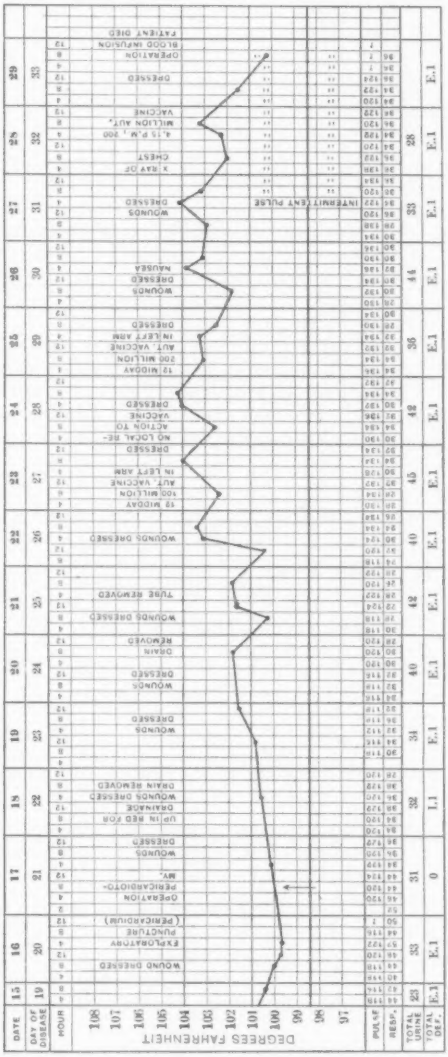
[1] Surgical Treatment of Suppurative Pericarditis, Tr. Am. Surg. Assn., 1897, xv, 101.
[2] Ann. Surg., 1900, xxxii, 769.
[3] Ibid., 1909, xlix, 60.
[4] Ibid., 1915, lxii, 660.

34; pulse, 105. He was put to bed and preparations made for possible pneumonia, which on the next day, December 31, gave frank signs at the lower right lobe. The blood count showed



hemoglobin, 85 per cent.; red blood cells, 4,900,000; white blood cells, 10,000. The differential count showed 85 per cent. polynuclears. Urine showed a specific gravity of 1.031, otherwise

negative. The temperature ranged between 104° and 105° for two days, falling to between 102° and 104° on the following two days, remaining at 102°, with one slight rise, for the following four days.



On January 8, the twelfth day of the disease, it was noted that the tubular quality of inspiration and expiration was more intense along a transverse line at the level of the angle of the scapula, with a

diminishing intensity toward the base of the lung. An exploratory puncture on this date revealed a thin, purulent exudate. Thoracotomy and drainage were performed immediately by one of us (Pool). Cultures from this exudate yielded Staphylococcus pyogenes aureus, from which vaccines were prepared. Patient did well for the next eight days, temperature gradually falling to 99.3°. On January 10 the following note was made: "Heart action strong and regular, no dropped beats. The rhythm is clock-like, there being no rest periods." On January 11 the following nurse's note: "Respirations labored most of the day. Pulse feeble and intermittent. Breathing better upon loosening dressing." January 15 and 16 showed a distinct change in pulse, somewhat irregular and weaker. On January 16 the color of the skin was noted as bad and the respirations labored. Physical signs suggested pericardial effusion, which was confirmed by exploratory puncture; a thin, purulent exudate being obtained, which on culture (by Dr. Mortimer Warren) revealed the same organism as that found in the pleura. The opening and draining of the pericardium was now considered as offering the only chance of relieving the severe cardiac embarrassment. This was done under local anesthesia by one of us (Pool) by the method described in the discussion below. While the patient's condition was critical during the operation, his color and pulse improved markedly the moment the pericardium was relieved of the purulent effusion. He continued to do well for six days following the operation, the pulse, however, being somewhat irregular and at no time forcible. The respirations occasionally became labored, but were always relieved after dressing the pericardial wound. On January 22 the calf of the right leg became painful and swollen, and it was evident on examination that phlebitis had developed. Blood culture at this time showed Staphylococcus aureus. On the evening of the 22d the heart action and breathing became greatly embarrassed, and the following note was made: "Temperature has steadily risen since operation; pulse weak; no heart sounds audible; drainage poor, Digital examination into the wound showed heart sealed to pericardium. Finger introduced deeply in wound, and fresh adhesions broken up, yielding a copious discharge of very thick stringy material and much pus. Patient's condition markedly improved and the heart sound became distinctly audible." On January 23 and 24 respiration and heart action remained good, and the drainage from the pericardium free. On January 26 the pulse became small and intermittent and there was considerable nausea. On January 27 the temperature reached $104\frac{2}{5}°$, falling to 102° on January 28. On this date the chest was radiographed. The report of the findings is as follows:

Indications of fluid and some air in the right pleural sac, with partial collapse of the lung. The heart shadow is distinctly enlarged, suggesting fluid in the pericardium.

Autogenous vaccines were employed, but it was realized that these were used too late in the disease to markedly influence its course. On January 29 the patient died.

PARACENTESIS PERICARDII AS A DIAGNOSTIC ADJUVANT. Although this procedure exposes to the danger of accidental injury to the heart, coronary artery or vein, pleura, lung or internal mammary artery, it must be emphasized that serious accidents have occurred so infrequently that there should be no hesitancy in performing paracentesis when the operation seems definitely indicated. The limitations of exploratory puncture should be definitely appreciated. Even when fluid is present, puncture has often proved unsuccessful. A dry tap may result from the needle becoming blocked with fibrin or with tissue that has been penetrated during its introduction. Moreover, the exudate, especially early, may lie in such a position as to be out of reach of the needle. If suppurative pericarditis is suspected, diagnostic punctures should not be made repeatedly; when several attempts have been made, puncture should be abandoned in favor of pericardiotomy. It must be stated that a number of writers decry the use of puncture for diagnostic purposes. Rhodes states that it can be of positive danger and is usually superfluous.[5]

Opinions differ widely as to the point of election in paracentesis of the pericardium. The favorite sites for punctures fall into two groups: those which are planned to avoid the pleura and those which disregard the pleura, but which are planned to avoid the heart. Under normal conditions the pleura can be avoided by introducing the needle in the region of the sixth and seventh cartilages of the left side, close to the sternum or below the tip of the xiphoid. On the other hand, cardiac injury is best avoided by puncture at a point situated somewhat internal to the lower part of the left limit of pericardial dulness. Curschman advocates this site in preference to one in the immediate vicinity of the cardiac margin or apex, because the effusion is very frequently associated with an enlarged heart, in which case the heart may extend beyond the apparent position of the apex; moreover, it is at times adherent to parietal pericardi. The disadvantage of the outer site is that the pleural cavity may be entered, a feature which is especially disadvantageous in the presence of suppurative pericarditis, but penetration of the pleural cavity is not a necessary complication, because the pleural edges are separated by the distended pericardium, and not infrequently the pleural layers are agglutinated to a considerable degree by adhesions, causing obliteration of the anterior part of the pleural sac.

The following sites are most suitable for paracentesis of the pericardium:

[5] Some features in this article are taken, with permission of the publishers, from Johnson's Operative Therapeusis: Heart and Pericardium, by E. H. Pool, published by D. Appleton & Co.

1. A point slightly internal to the left limit of dulness, in the fifth or sixth intercostal space.

2. Midline immediately below the xiphoid process. This situation is recommended by François and others as the point of election for entrance, because here the pericardium is reached by the shortest route without injury to bloodvessels, pleura, and peritoneum, while the exudate can be evacuated before the heart is touched. The only contra-indications of this method are extreme malformations of the sternum and a high degree of tympanites. Under local anesthesia a fine trocar or hollow needle of sufficient length is inserted and pushed directly upward to a distance of 2 cm. along the posterior surface of the xiphoid. In this way the peritoneum is avoided, and after passing through the diaphragm the point of the needle is directed upward and backward.

3. A point in the angle formed by the base of the xiphoid process and the seventh left cartilage at its insertion. The choice between these sites must depend upon individual indications.

Pericarditis occurs as a relatively frequent complication in a large number of diseases, the most frequent of which are inflammatory rheumatism, nephritis, pleurisy, pneumonia, pyogenic infections, and tuberculosis, but there are "few infectious diseases which may not implicate the pericardium." (MacPhedran.[6])

The clinical picture of pericarditis with effusion is variable. Besides the local objective signs, the cardinal sign of exudative pericarditis being a characteristic cardiac dulness which is at first increased upward, there are present to a variable degree, symptoms due to: (1) involvement of the heart muscle (myocarditis), which is the most frequent and serious lesion; (2) endocarditis (these two are especially frequent in association with rheumatism); (3) mechanical disturbance of the heart as a result of pressure exerted by the effusion; this, however, is infrequent; (4) general constitutional disturbances, depending upon the type of the infection, the pericardium offering a large surface for absorption; (5) an associated lesion or disease.

Suppurative pericarditis represents an abscess corresponding to a part or the whole of the pericardial sac. The most frequent organisms are the streptococcus, staphylococcus, and pneumococcus. The exudate is usually purulent from the beginning of the attack, although occasionally it develops in the course of a non-suppurative pericarditis. As a rule, purulent pericarditis is secondary in the course of a pyogenic infection, and under such conditions it frequently constitutes a "terminal infection;" in rare cases suppurative pericarditis is primary; in some cases the infection is introduced through a wound.

Suppurative pericarditis is characterized by the local sign of

[6] Osler's Modern Medicine, iv, 40.

pericarditis, with effusion and constitutional symptoms of a septic character. The fact that the pericarditis is frequently a secondary lesion causes its presence to be overlooked in many cases. In children the symptoms of pericarditis are particularly apt to be masked. On the other hand, in many cases in which the lesion has recognized and the pericardium drained, the operation has been unsuccessful because a coexisting purulent focus, especially empyema, has been overlooked. It follows that in septic processes it is necessary to watch for the development of pericarditis; moreover, the recognition of such a condition should not cause less thoroughness in the search for other foci.

The distribution of the fluid in the distended sac must be understood in order to appreciate the technical details of paracentesis or pericardiotomy. First, the recesses become filled, the fluid gathering chiefly in the region of the base of the heart; then the bulging pericardium, confined anteriorly, separates the yielding lungs and pushes the diaphragm downward. The increasing exudate progressively distends the pericardium, for the most part to the left and posteriorly It must be emphasized that the heart usually, though not invariably, occupies a low position and remains applied to the anterior wall of the thorax. Occasionally an inflammatory process may become localized by the formation of adhesions and the limitation of the exudate to a circumscribed part of the sac.

PERICARDIOTOMY. The methods of pericardiotomy may be divided for convenience into two groups:

1. Procedures which reach the pericardium through the thoracic wall.

2. Procedures which enter from below the costal arch, the epigastric route.

The first method of procedure includes:

Resection of the sixth cartilage, as recommended by Kocher, Axhausen, and Pels-Leusden.

Resection of the fifth cartilage, Gussenbauer and Ollier.

Resection of the fifth and sixth cartilages, Delmore and Mignon.

The second method of procedure has been accorded the preference by Larrey, Mintz, L. Rehn, and Allingham. The approach is in part or entirely from below the costal arch, and is both extra pleural and extraperitoneal.

PERICARDIOTOMY FOR THE EVACUATION OF A PURULENT EXUDATE. In the treatment of suppurative pericarditis the method of pericardiotomy to be efficient must provide for satisfactory drainage, and be sufficiently simple to be rapidly performed under local anesthesia if necessary. Adequate drainage is the essential feature, and upon this must depend, to a large extent, the choice of method. In the interest of good drainage it is important (1) to open the pericardium at its lowest point, and (2) to provide such an opening as will ensure ready egress for accumulation of fluid in both the

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right and left spaces of the pericardial sac. Local anesthesia is frequently imperative because the heart is apt to be dilated and insufficient as a result of endocarditis and myocarditis. It is necessary that the work be done with the least possible associated injury. It is not sufficient to avoid wounding the heart; if possible the heart should not be touched. Further, thorough anesthetization of the pericardium is advisable on the basis of significant experimental work, which we will briefly summarize.

Serious disturbances of the heart action have been noted repeatedly to result from irritation of the pericardium. Heitler's[7] observations on dogs showed that irregularity of the heart follows mechanical or electrical stimulation of the pericardium. D'Agata[8] (1912) in animal experiments found that a sudden drop in the blood-pressure occurred as a result of grasping and incising the pericardium. These phenomena are presumably reflex in character; they help to explain analogous clinical observations. Thus, Harrigan[9] reported temporary arrest of the heart upon incision of the pericardium in a case of suppurative pericarditis.

Heitler and D'Agata found in their experiments that preliminary cocainization of the parietal layer of the pericardium prevented the occurrence of irregularity of the heart and the lowering of the blood-pressure. Heitler recommended that before an incision is made in the pericardium it should be anesthetized by means of a 10 per cent. solution of cocain applied to its surface. Presumably novocain by injection could be substituted to advantage in man.

The two methods most appropriate for drainage in suppurative pericarditis will be described in detail.

1. Resection of the sixth costal cartilage. (Kocher.[10])
2. Resection of the seventh, or sixth and seventh costal cartilages (Rehn.)

Resection of Sixth Cartilage. This method affords adequate drainage at a dependent part of the sac, is simple and quick of execution, may be performed readily under local anesthesia, and the exposure may be extended easily in any direction. It appears the best method for general use and was employed in our case.

With the patient in a semirecumbent position, an incision is made in the course of the sixth cartilage and rib, passing from the midline obliquely outward. The perichondrium is incised in the direction of the wound and separated. The cartilage is then severed close to the sternum and lifted, the perichondrium being separated from its posterior surface. The cartilage is broken at its junction with the rib and removed. The internal mammary artery should be divided between ligatures when exposed. The triangularis sterni is then split. An effort should be made to identify the anterior

[7] Med. Klin., 1910, vi, 974.
[8] Arch. f. klin. Chir., 1912, xcviii, 460.
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edge of the underlying pleura, which should be pushed outward. The pericardium is thus exposed. It should be grasped and lifted with two pairs of toothed forceps and incised. (The importance of anesthetizing the pericardium has been emphasized above.) The pericardial incision is extended with blunt-pointed scissors. The pus should be allowed to escape slowly. A finger is then introduced to break up adhesions and to evacuate walled-off accumulations of pus. Should the access be too small to allow separation of adhesions, or for adequate drainage, the opening may be enlarged in an appropriate direction by resection of part of the sternum, excision of part of the sixth rib, or removal of the fifth or the seventh costal cartilates. Residual fluid should be removed by aspiration, as in abdominal operations, and sponging or wiping of the serous surfaces should be reduced to a minimum. Clots of fibrin should be extracted with forceps. Irrigation with sterile salt solution has been used for the removal of clots of fibrin, but is not to be recommended. The use of antiseptic fluids is contra-indicated. In closing the wound it is advisable, especially if the pericardium is considerably separated from the sternum, to suture it to the skin. This not only facilitates drainage but "diminishes the risk of contamination of the anterior mediastinum." (Eliot.)

Drainage is favored by the movements of the heart, which in the absence of adhesions tends to force out any accumulated pus (Eliot). The kind of drain which should be used is a detail which demands some discussion. The choice lies between a rubber tube and soft drain. Tubes afford the most satisfactory drainage and may be sutured to the soft parts, so that they are not readily displaced. But if the tube should come into contact with the heart it is likely to interfere with the cardiac action, as occurred in a case operated upon by Riedel.[11] Rehn[12] recommends two rubber tubes, one passing to the right side and one to the left to drain both pockets. Eliot, Riedel and others favor soft drains. A general rule cannot be made, as the mode of drainage must be such as to fit the individual case.

Two cigarette drains were employed in our case. They did not drain satisfactorily. We are of the opinion that split soft rubber tubes would have been better.

The sitting position should be enforced in the after-treatment as most favorable for drainage. The wound heals, as a rule, without a persistent sinus, and "after healing is complete, insufficient or irregular heart action is the marked exception; neither is there any indication of cardiac displacement." (Eliot.)

Resection of the Seventh or Sixth and Seventh Costal Cartilages. A curved incision of about 6 cm. is made along the lower margin

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of the seventh left cartilage to the base of the ensiform process, across which the incision is continued transversely. The incision is deepened and the seventh costal cratilage divided. The internal mammary artery is preserved. Detaching the soft parts, the finger is passed under the sternum, between the lower portions of the triangularis sterni muscle and the sternal portions of the diaphragm. A piece of the sternum and seventh costal cartilage is removed, followed by resection of the sixth costal cartilage if necessary. The pericardium is readily reached without opening the pleura. The more distended the pericardium the easier the operation. (Description after Kuttner.[13])

PROGNOSIS. Suppurative pericarditis demands immediate incision and drainage. The prognosis in purulent pericarditis is in general unfavorable, yet in suppurative processes which are confined to the pericardium early operation is quite often followed by recovery; on the other hand, when the pericardium is involved secondarily in the course of a general sepsis, recovery is infrequent. Nevertheless, cases that appear hopeless are occasionally saved by operation. As mentioned below a prognosis must depend largely on the characteristics of the organism causing the disease, but as yet this has not been studied with sufficient accuracy to consider it in this report.

CONCLUSION. The results of pericardiotomy are necessarily modified by the gravity of the fundamental disease, but striking cures have followed operation in apparently hopeless cases of severe infection. In studying our case, together with the literature here quoted, the need for a bacteriological division of these cases seems imperative. From Rufus Cole's work we now have a new aspect of the clinical condition known as pneumonia. This new view-point is bacteriological in character. We now know that the anatomical diagnosis of consolidation of a lobe, or of a purulent accumulation in the pleura or pericardium may be correct, and yet the clinical course vastly different according to the organism producing the condition. In traumatic injuries to the pericardium when operation has been successfully performed and recovery has followed, the favorable factor is probably the absence of bacterial infection. In our case a most virulent organism, the staphylococcus, was the cause of a bacteriemia. Was the dose and virulence of the toxin so overwhelming that no known method would have been sufficient to overcome these? Perhaps vaccines should have been used from the time of the furunculosis, but at that time no culture was taken and there was prompt recovery from a single abscess in the neck. The question naturally arises as to whether we should regard these simple abscesses as demanding immediate vaccine treatment as a routine, fearing the development of larger areas of pus in more vital localities.

[13] Bier-Braun-Kummell, Chirurg. Operationslehre, 1912, ii, 88.

Is pericardiotomy justifiable or imperative in all cases of suppurative pericarditis? As to the justification for pericardiotomy in all cases of suppurative pericarditis it would seem that the operation should be performed as an emergency procedure even in desperate cases. The importance of an early diagnosis cannot be too strongly urged. Whether exploratory puncture reveals a purulent or a non-purulent effusion, cultures should be made and an accurate report presented. We may expect that further bacteriological studies will throw light upon the general clinical course due to different types of organisms. Thus the prognosis in a given case may be made more exact and the indications for surgical intervention may be further developed. This case and those quoted from literature go to emphasize the fact now being grasped by the profession that pneumonia is a local manifestation of a systemic infection, and in dealing successfully with it measures capable of reaching the antigen power of the system must be sought and employed.

THE FOLLOWING CASES FROM LITERATURE ARE ARRANGED AS FOLLOWS:

Suppurative pericarditis following: (1) pneumonia; (*a*) lobar; (*b*) broncho. (2) Empyema.

A bacteriological division is not possible, as cases have not been studied accurately with this feature especially considered. This is a most important aspect of suppurative pericarditis and offers a field for future investigation. From the data in some of the following reports it is difficult to determine whether pneumonia was clinically present.

1. Pneumonia: (*a*) lobar.

Female, aged forty years. Croupous pneumonia, empyema, and purulent pericarditis. Thoracotomy for empyema; six days later pericardiotomy, Ollier's method. Fifth rib resected. Pericardium opened 400 to 450 c.c. of pus with fibrinopurulent exudate evacuated. Drainage tube introduced and held by suture. After operation patient in collapse and cyanotic. Gradual recovery. (Original article not accessible.) (Tallquist; cf. Rhodes.[14])

Boy, aged twelve years. Bilateral diplococcus pneumonia. Ill for about two weeks and was in bad condition before the operation, heart dulness extending two fingers' width beyond the right sternal margin and to the midaxillary line on the left side. Incision along the lower margin of the seventh costal cartilage. A skin and cartilage flap formed which was turned upward, permitting access to the space through which the pericardium could be opened without injuring the pleura; 100 c.c. of seropurulent fluid was evacuated, and a tampon introduced. General condition continued to improve;

[14] Finska laksallsk. Handl. Helsingfors I, 1912, 575–595.

of the seventh left cartilage to the base of the ensiform process, across which the incision is continued transversely. The incision is deepened and the seventh costal cratilage divided. The internal mammary artery is preserved. Detaching the soft parts, the finger is passed under the sternum, between the lower portions of the triangularis sterni muscle and the sternal portions of the diaphragm. A piece of the sternum and seventh costal cartilage is removed, followed by resection of the sixth costal cartilage if necessary. The pericardium is readily reached without opening the pleura. The more distended the pericardium the easier the operation. (Description after Kuttner.[13])

PROGNOSIS. Suppurative pericarditis demands immediate incision and drainage. The prognosis in purulent pericarditis is in general unfavorable, yet in suppurative processes which are confined to the pericardium early operation is quite often followed by recovery; on the other hand, when the pericardium is involved secondarily in the course of a general sepsis, recovery is infrequent. Nevertheless, cases that appear hopeless are occasionally saved by operation. As mentioned below a prognosis must depend largely on the characteristics of the organism causing the disease, but as yet this has not been studied with sufficient accuracy to consider it in this report.

CONCLUSION. The results of pericardiotomy are necessarily modified by the gravity of the fundamental disease, but striking cures have followed operation in apparently hopeless cases of severe infection. In studying our case, together with the literature here quoted, the need for a bacteriological division of these cases seems imperative. From Rufus Cole's work we now have a new aspect of the clinical condition known as pneumonia. This new view-point is bacteriological in character. We now know that the anatomical diagnosis of consolidation of a lobe, or of a purulent accumulation in the pleura or pericardium may be correct, and yet the clinical course vastly different according to the organism producing the condition. In traumatic injuries to the pericardium when operation has been successfully performed and recovery has followed, the favorable factor is probably the absence of bacterial infection. In our case a most virulent organism, the staphylococcus, was the cause of a bacteriemia. Was the dose and virulence of the toxin so overwhelming that no known method would have been sufficient to overcome these? Perhaps vaccines should have been used from the time of the furunculosis, but at that time no culture was taken and there was prompt recovery from a single abscess in the neck. The question naturally arises as to whether we should regard these simple abscesses as demanding immediate vaccine treatment as a routine, fearing the development of larger areas of pus in more vital localities.

[13] Bier-Braun-Kummell, Chirurg. Operationslehre, 1912, ii, 88.

Is pericardiotomy justifiable or imperative in all cases of suppurative pericarditis? As to the justification for pericardiotomy in all cases of suppurative pericarditis it would seem that the operation should be performed as an emergency procedure even in desperate cases. The importance of an early diagnosis cannot be too strongly urged. Whether exploratory puncture reveals a purulent or a non-purulent effusion, cultures should be made and an accurate report presented. We may expect that further bacteriological studies will throw light upon the general clinical course due to different types of organisms. Thus the prognosis in a given case may be made more exact and the indications for surgical intervention may be further developed. This case and those quoted from literature go to emphasize the fact now being grasped by the profession that pneumonia is a local manifestation of a systemic infection, and in dealing successfully with it measures capable of reaching the antigen power of the system must be sought and employed.

THE FOLLOWING CASES FROM LITERATURE ARE ARRANGED AS FOLLOWS:

Suppurative pericarditis following: (1) pneumonia; (*a*) lobar; (*b*) broncho. (2) Empyema.

A bacteriological division is not possible, as cases have not been studied accurately with this feature especially considered. This is a most important aspect of suppurative pericarditis and offers a field for future investigation. From the data in some of the following reports it is difficult to determine whether pneumonia was clinically present.

1. Pneumonia: (*a*) lobar.

Female, aged forty years. Croupous pneumonia, empyema, and purulent pericarditis. Thoracotomy for empyema; six days later pericardiotomy, Ollier's method. Fifth rib resected. Pericardium opened 400 to 450 c.c. of pus with fibrinopurulent exudate evacuated. Drainage tube introduced and held by suture. After operation patient in collapse and cyanotic. Gradual recovery. (Original article not accessible.) (Tallquist; cf. Rhodes.[14])

Boy, aged twelve years. Bilateral diplococcus pneumonia. Ill for about two weeks and was in bad condition before the operation, heart dulness extending two fingers' width beyond the right sternal margin and to the midaxillary line on the left side. Incision along the lower margin of the seventh costal cartilage. A skin and cartilage flap formed which was turned upward, permitting access to the space through which the pericardium could be opened without injuring the pleura; 100 c.c. of seropurulent fluid was evacuated, and a tampon introduced. General condition continued to improve;

[14] Finska laksallsk. Handl. Helsingfors I, 1912, 575–595.

patient discharged after about two months, with normal heart boundaries and without any disturbance except a fistula from which a small amount of pus escaped. (Mintz.[15])

Boy, aged five years. Bilateral croupous pneumonia. Apex beat could not be felt; the heart sounds were very faintly audible. Dulness two fingers' width beyond the right sternal margin and a finger's width beyond the nipple-line to the left side. Exploratory puncture in the fifth intercostal space yielded 60 cm. of thick fluid pus, and was followed by visible improvement. Pericardiotomy, with resection of the fifth costal cartilage, according to Ollier. Half liter of pus escaped. The heart was found lying behind and above, floating free in the large cavity. Subperichondral resection of the fourth costal cartilage was added. Drainage with rubber tube the thickness of a thumb. In three weeks cavity had entirely disappeared without leaving a thoracic deformity. Good recovery.

The author emphasizes the advantages of pericardiotomy with costal resection on the basis of Terrier's statistics, and recommends early operative interference, which has a simple technic as in empyema and yields the best results as to life and rapid healing. (Strauss.[16])

Pericarditis secondary to double pneumonia. The pericardium was opened in the midaxillary line and a large amount of pus was evacuated. A few days after the operation there were signs of pus farther back, and an incision there revealed an empyema. The patient died. (Blake.[17])

Boy, aged nine years. Left-sided pneumonia. On the ninth day dulness was noted, extending on the right side one finger's breadth beyond the right sternal margin; on the left side two fingers' width beyond the left maxillary line. Heart sounds inaudible; superficial veins of thorax much distended, especially on right toward shoulder. Pericardiotomy was performed by v. Eiselberg, in spite of the patient's apparently moribund condition. Resection of fifth costal cartilage. Exploratory puncture of bulging pericardial sac yielded pus. Transverse incision of pericardium; escape of about one liter of thick yellow pus. Two rubber drainage tubes. Pulse was plainly palpable three hours after operation. General condition rapidly improved. A fistula persisted for nine weeks. Recovery. Bacteriological examination of the pus showed streptococci. The patient was reëxamined nine years after operation and was found to be in good condition. (Walzel.[18])

Man, aged thirty-one years. Pneumonia chiefly involving the

[15] Chirurgia, April, 1912, vol. xxxi (Russian); Centralblatt f. Chir., 1912, No. 30, p. 1046.

[16] Nuremberg Med. Soc., meeting of July 15, 1909; München. med. Wchnschr., 1909, No. 39, p. 2039 (or 7).

[17] New York Surgical Society, October 28, 1908; Annals of Surgery, 1909, xlix, 142.

[18] Mittlg. a. d. Grenzgeb. d. Med. u. Chir., 1913, xxv, 264.

left lung. Three weeks later cardiac dulness four fingers' breadth to the right of the sternum, with muffled heart sounds, persistent high temperature, considerable cyanosis, and dyspnea. Pericardium was incised, after removal of the seventh costal cartilage and a portion of the sixth; 3 to 4 ounces of fluid were withdrawn. Numerous adhesions between the right heart and parietal pericardium were broken down and a tube was inserted, which was removed on the sixth day after the operation. The patient made a good recovery. (Pandlebury; cf. Eliot.[19])

Male, aged thirty-six years. Bilateral pneumonia. On the thirteenth day the pericardium was considered involved. Twenty-first day the cardiac dulness was decidedly increased in all directions, especially toward the right. Exploratory puncture yielded a turbid greenish fluid. On culture a pure growth of pneumococci. Incision of pericardium and evacuation of about a pint of pus. Examination showed pneumococci. Drainage tube removed twenty-six days after operation and the patient left the hospital fifty-one days after operation, free from physical signs of an adhesive pericarditis. (Scott; Eliot.[20])

Woman, aged thirty-eight years. Puerperal sepsis and double pneumonia, three weeks before admission to the hospital. Extensive area of cardiac dulness, the heart sounds being almost inaudible. Puncture of the pericardium in the fourth intercostal space yielded pus. Excision of the fourth left costal cartilage with evacuation of 20 ounces of grayish-yellow pus. Drainage, with profuse discharge, until death one week after the operation, probably due to suppurative peritonitis.

Examination of the pus from the pericardium showed various streptococci and staphylococci. (Hall; Eliot.[21])

Patient, aged two and a half years. Pneumonia a month before admission to the hospital, when the diagnosis was made of a probable empyema at the right base, with a purulent pericarditis. Chloroform anesthesia. Oblique incision exposing the fifth left cartilage, which was removed in part with adjacent part of sternum. Incision of the pericardium with evacuation of about half a pint of thin greenish-yellow pus under pressure. A rubber drainage tube was inserted. Pericardial fluid contained a coccus which resembled pneumococcus. Temporary improvement. The empyema pus, obtained by operation on next day, showed a coccus which resembled the pneumococcus; other forms of cocci and bacilli were also numerous. The patient died in the sixteenth week after the operations.

Necropsy. On opening the thorax there was found a condition of universal dense adhesion of the contained viscera to each other and

[19] Lancet, October 22, 1904, p. 1144.
[20] New York Med. Jour., January 30, 1904, p. 198.
[21] Lancet, 1903, ii, 951.

to the chest wall, and the whole anterior mediastinum was occupied by a thick fibrogelatinous deposit. The wound over the pericardial area drained a small pocket on the anterior surface of the heart, but elsewhere the pericardium was inseparably adherent to the heart in front. On attempting to separate the pericardium from the left lung an abscess cavity was opened, with a discharge of 3 ounces of pale thick pus. This cavity lay behind, and to the left of the heart and within the pericardial sac; its walls were very thick from the presence of fibrinous matter similar to that occupying the mediastinum. Between the heart and the right lung there lay another smaller cavity, independent of the first, and full of inspissated pus. The pus in both cavities was dry and gritty to the feel, as if impregnated with lime salts. The tracheal glands were greatly enlarged, but free from all sign of tubercle. The pleuræ were everywhere adherent. (Coutts and Rowland; Eliot[22].)

Boy, aged sixteen years; sore throat, followed a day or two later by most of the symptoms of pneumonia of the left base without very distinct physical signs. At the end of three weeks, a left empyema was opened, without much relief to the general symptoms. Ten days afterward, the pericardium was opened, with only slight temporary general improvement. Death thirteen days after incision of pericardium, apparently due to external hemorrhage from the heart through rupture of a small parietal abscess.

Bacteriological examination proved the presence of pneumococci in great numbers in the blood, death being the result of a general pneumococcus pyemia. (Sibley, Arbuthnot Land and Rowell; Eliot.[23])

Pericarditis developed after a severe (left-sided) pneumonia. Aspiration showed the presence of a purulent effusion. Thirty ounces of the fluid were removed by aspiration. Pericardiotomy under local anesthesia, nearly 40 ounces evacuated. The pleura was slightly incised at the operation, and probably became infected, although it was at once carefully closed. The patient's condition at once improved and the alarming acute symptoms disappeared. The right pleura extended beyond the sternum, owing to the compensatory hypertrophy of the right lung, because of the pneumonia in the left. The accidental incision of pleura probably followed by infection. Patient died of purulent pneumonia a few days later. (Connor, L. A.; operator, Stimson.[24])

Male, aged fifteen years. Severe double lobar pneumonia. Pericardial friction sound heard on the fourteenth day. No increase of heart dulness could be demonstrated for two days, however. Ten days later the patient was in very bad condition and was aspir-

[22] Brit. Med. Jour., 1904, i, 9.
[23] Ibid., May 23, 1903, p. 1192.
[24] Med. News, January 20, 1900, lxxvi, 115; New York Academy of Medicine, stated meeting, December 19, 1899.

ated for the pericarditis, with removal of 18½ ounces of pus. This was found to contain a pure culture of the pneumococcus. No improvement. Operation under local anesthesia, incision from third to fifth intercostal space; resection of three-quarters of an inch of the costal cartilages. Penetration of pericardium was at once followed by a tremendous gust of pus, estimated at 50 to 60 ounces. Wound was left open; no artificial drainage. Irrigations with normal salt solution. Patient in excellent general condition three months after the operation. Bacteriological examination showed pneumococcus in pus in pure caulture; pneumococcus and streptococcus in sputum. (Lilienthal, H.[25])

Male, aged twenty-four years. Lobar pneumonia. Sudden attack of chills and fever; followed by dyspnea and pain in left half of chest. Exploratory puncture. Incision in middle of fifth intercostal space, between sternum and nipple. Incision showed the existence of pleural adhesions. The pericardium was opened, and a large stream of pus spurted out. Patient was in collapse, but recovered after digital massag of the heart. The fluid had a greenish color, contained some fibrin flakes, and amounted to about 1½ liters. A thick long drainage tube was inserted, reaching to the back portion of the pericardium. After a fortnight, careful irrigations of the pericardial cavity with salt solution were begun and repeated every third day, to wash away the fibrin coagula, but it finally became necessary to do a resection of the fifth rib, in order to obtain a freer access to the pericardium and break up adhesions. After this, recovery was rapid; fistula closed a month later. Patient discharged in good condition, about five months after admission to the hospital.

No bacteriological growth from original fluid; microscope showed small, short bacilli often lying two or three together. Bacillus pyocyaneus grew in all cultures made from the discharge after operation. (Ljunggren, C. A.[26])

Male, aged twenty-two years. Acute pneumonia. Pericardiocentesis; incision in fourth left interspace, under cocain. Forty-five ounces of pus escaped. Irrigation with boracic acid. Tube drain. Temporary improvement was followed by purulent effusion in both pleural cavities. Bilateral costal resection and escape of 35 ounces of pus. Pneumococci were present in the pus from both pleural cavities. The pus from the pericardium showed almost a pure culture of pneumococci. Death one month after admission to the hospital. (Sevestre, R.[27])

Male, aged twenty-two years. Purulent pericarditis due to pneumonia. No pericardial friction. Double empyema. Aspiration fourth space one inch from sternum; incision in same place. Evacu-

[25] Med. News, 1899, lxxv, 697. Presentation of case, New York Academy of Medicine, Section on Surgery, November 13, 1899.

[26] Nord. Med. Arkiv., 1898, ix, 1.

[27] Lancet, April 23, 1898, i, 1109.

ation of large amount of pus. Death eighteen days after operation. Autopsy findings: Thickened pericardium; double empyema. (Bradbury, quoted by Sevestre.[28])

Boy, aged fourteen years. Pleuritis, pneumonia. Abscess of thigh, acute arthritis of left shoulder. There was much dyspnea and some cyanosis, and there were physical signs of pneumonia of the base. The left pleura was tapped, but only a few ounces of serum were evacuated; from the pericardium 24 ounces of pus was drawn off. Though relieved for a time the dyspnea and cyanosis returned, and the patient died. (West, S.[29])

Boy, aged six years. Pyemia and septic pneumonia after necrosis of metatarsus. Pus having been shown by aspiration, the fifth interspace was incised and 8 ounces of pus evacuated, no irrigation. Drainage tube of $3\frac{1}{2}$ inches. Temporary relief, but rise of temperature on next day (104° F.). Patient lived seven days after the operation. (Davidson, P.[30])

The author says that this case resembles Bronner's observation in several points, viz., in the occurrence together of pleurisy, pneumonia, and pyopericarditis, also in the character of the pneumonia, sometimes called "wandering pneumonia;" and in the rise of temperature after the opening of the pericardium.

Boy, aged six and three-quarter years. Admitted with history of a fortnight's illness, with cough, fever and pain in the left side. Pus was obtained on exploratory puncture in left pleural cavity. Next day, thoracotomy and removal of 10 ounces of pus. Drainage. Increasing precordial dulness. Exploration of pericardium yielded pus. Incision in fourth interspace; escape of several ounces of creamy pus. Tube drain. Pericardium and empyema took about five weeks to heal. Patient was discharged in good condition two months after admission.

Girl, aged eleven years. Influenza, pneumonia, and empyema. Thoracotomy of right pleura pericardiotomy. Incision in fourth space one inch from sternum; escape of 2 pints of thick pus. Irrigation: short tube drain. Pericardial cavity was syringed out on eleventh day after operation. Death twenty-six days after the incision into the pericardium, on the sixtieth day of illness. No bacteriological examination of pus. Bronner says that the pneumonia resembled Ribbert's streptococcus pneumonia, characterized by great irregularity of the percutory and auscultatory symptoms, by a tendency to spread, and by irregular infiltration of the lung tissue on postmortem examination. (Bronner; operator, Teale.[31])

Female, aged twenty-two years. Right-sided pneumonia and right empyema pericardiocentesis. Seven days later pericardio-

[28] Lancet, 1898, i, 1111.
[29] Brit. Med. Jour., December 8, 1883, p. 1129; February 21, 1891, p. 404.
[30] Ibid., March 14, 1891, p. 578.
[31] Ibid., February 14, 1891, p. 350.

tomy; incision in third space near sternum. Escape of large amount of purulent fluid. Drainage. No irrigation. Left empyema after operation. Death (pyemia) on thirteenth day after pericardiotomy. Bacteriological findings: Bacilli in pus, small short ends, neither pneumococci nor streptococci. Autopsy findings showed pus in pericardial and both pleural cavities, as well as acute nephritic changes. (Sievers, R.[32])

Male, aged twenty years. Right lobar pneumonia; pleural and pericardial effusion. Aspiration. Incision in fourth intercostal space, between nipple and sternum, on thirty-fourth day. No irrigation. Tube drain, free outflow of pus. Death from heart failure in thirty days, after pericardiotomy, and sixty-fourth day of illness. Bacteriological findings: Fraenkel's diplococcus in pus discharged from wound. (O'Carroll, J.[33])

Male, aged thirty-three years. Influenza pleuropneumonia followed by purulent pericarditis. Tapped three times. Incision in third intercostal space; pus under high pressure escaped on incision of pericardium. Local anesthesia (Schleich). Irrigation, boric acid, gauze drain. Immediate relief and improvement. Recovery in four and a half weeks. Discharged from hospital three months after the pericardiotomy. No bacteriological examination of pus. (Bohn, H.[34])

Male, aged twenty-six years. Pneumonia. Pericardial effusion. Left empyema. Pericardiocentesis; incision in fifth left space $1\frac{1}{2}$ inches to left of sternum. Large amount of pus evacuated. Irrigation with salt solution. Drainage with two tubes. Resection of fifth rib, six days later, for better drainage. Empyema; pneumothorax after operation. Recovery. Bacteriological findings: Pneumococcus in pus from pericardium and empyema. (Shattuck, F. C.; operator, Porter, C. B.[35])

Girl, aged twelve years. Influenza and pleuropneumonia. Paracentesis of pericardium. Resection of fifth and sixth ribs; pleura opened and sutured after evacuation of some bloody serum. Irrigation with boric acid solution; drainage, two large tubes. Recovery in six weeks. No bacteriological examination of pus. (Björkman; quoted by Roberts.[36])

(*b*) Broncho:

Boy, aged ten years. Twelve days before admission he fell forcibly upon the left anterior part of his chest. Three days later felt sick and complained of pain in the left side.

Examination. Fever, cough, dyspnea. Temperature, 39°; pulse, 130; respirations, 36; rather diaphragmatic, and left side of thorax

[32] Ztschr. f. klin. Med., 1893, xxiii, 26.

[33] Dublin Jour. Med. Sc., 1896, cii, 11; Same case as that of O'Carroll in Tr. Royal Acad. Med., Ireland, 1896, xiv, 106.

[34] Deutsche med. Wchnschr., 1896, xxii, No. 48, 769.

[35] Boston Med. and Surg. Jour., 1897, cxxvi, 438.

[36] Am. Jour. Med. Sc., 1897, cxiv, 662.

remaining immobile in respiration. Dulness on left from clavicle embracing, below, the precordial area, and extending one finger's breadth beyond right sternal line. On the left the dulness continues to axillary line. Signs of fluid in left pleura. Bronchopneumonia and congestion.

Diagnosis. Pleuropericarditis. Aspiration in seventh space, axillary line. No result. Condition gradually worse, with abdominal and precordial pain, loss of sleep from pain and cough. Streptococci in expectoration. Nine days after entrance into hospital an aspiration through fifth left intercostal space in mammary line; pus withdrawn. Operation. Incision 4 cm. long in fifth intercostal space, commencing 1 cm. from left sternal margin. Down to pericardium. Aspirated 250 grams of pus. Cultures showed white colonies, Gram-positive. Rapid fall in temperature.

Patient had a stormy time with the bronchopneumonia, but left the hospital with normal heart outlines and good action. No retraction of intercostal spaces. (Björkman; Imerwol; cf. Rhodes.[37])

Boy, aged eight years. Admitted as a case of appendicitis. Tentative diagnosis was bronchopneumonia, affecting principally the left lung, with possible pericardial effusion. Three days later pericardial paracentesis; 6½ ounces of thick purulent fluid withdrawn, giving almost immediate relief. Two days later pericardiotomy, after resection of part of fifth rib about 1½ pints of pus evacuated. Drainage tube inserted; discharge free. After first gaining some strength, the patient slowly lost ground, under symptoms of pyemia. Death. Examinations of the purulent discharge showed a double coccus (thought not to be pneumococcus by laboratory interne). Examination of the sputum for tubercle bacilli and pneumococci was made with negative results. According to the postmortem report, the left lung was totally collapsed and compressed to a very small size into the apex of the left chest. In the base of the right lung there was a cavity about the size of a walnut, containing bloody purulent fluid. No communication between the pericardium and lungs or pleura. The collapsed left lung was explained by the fact that the pleura was probably accidentally opened during the operation. (Gengenbach, F. P.; cf. Eliot.[38])

Boy, aged four years. Severe bronchopneumonia. Pericardiotomy. Resection of fifth cartilage; pleura not opened. Seropurulent fluid and typical pus from deepest part of sac, about 10 ounces. No irrigation; gauze drain. Death in three days. There was no evidence of, and no history to suggest that the whole condition was pyemia. The pus from the pericardium gave on cultivation an almost pure growth of Friedländer's pneumococcus. (Robinson, H. B.; Case II.[39])

[37] Hygeia, 1896, lviii, 189; Am. Surg., 1915, lxii, 660.
[38] Colorado Med., 1906, iii, 198. (A case of purulent pericarditis.)
[39] Brit. Med. Jour., November 26, 1898, ii, 1605.

2. Empyema.

Male, aged twenty-eight years. About January 7, 1913, when working, he developed pain in the right side, shortness of breath, cough, and tenacious sputum, and came to hospital for treatment. Temperature, 102.4°; right lower lobe dull. Crepitant rales and bronchial breathing.

January 14, complained of pain in right side, also pain in precordial region. Pericardial friction rub heard.

January 31, needle introduced in sixth interspace just beyond apex of heart and a quart of pus drawn off.

Operation. Ransohoff. Local anesthesia (cocain). Sixth rib, 2 inches or rib resected, starting one-half inch from sternum. Pericardium punctured and pus drawn off. Pericardium opened liberating about 3 pints of a fairly thick yellowish pus. Pericardium stitched to thoracic wall and gutta-percha drain inserted.

March 5, patient died.

Autopsy. Right lung showed congestion and an infarct in lower portion of middle lobe; pleura normal. Left pleural cavity contained about a quart of greenish pus, and the lung was found compressed into upper part of pleural cavity. It showed infarction and congestion.

Pericardial sac was entirely obliterated except posteriorly below and anteriorly where drainage had been established.

Pancreas, spleen and alimentary canal normal.

Left kidney showed a total infarction due to thrombosis of renal artery. Right kidney was congested. (Ransohoff; cf. Rhodes.[40])

Boy, aged nine years. Two weeks before admission had chills and fever, pain in left side of thorax, fever high.

Diagnosis. Exudative pericarditis and pleurisy.

May 5, aspiration in axillary region, fifth interspace, gave pus from left plura.

May 6, aspiration in anterior axillary line, fifth left interspace 300 grams, seropurulent fluid. Aspiration of pericardium, fifth left interspace 3½ cm. from left sternal border gave pus. Improvement.

May 19, thoracotomy in fifth left space, axillary line; 350 grams of pus, drainage tube.

May 20, discovered that the pericardium opens into the left pleura by an orifice. Heart sounds audible and apex beat visible. For the next four days this orifice increased in size. Case gradually improved and discharge lessened. Drains removed July 15 and gauze substituted. Wound cicatrized.

July 20, no retraction of intercostal spaces. Normal. Pus showed pneumococci. Imerwol, V.; cf. Rhodes.[41])

Girl, aged six years. Empyema; thoracotomy. Pericardiotomy. Incision one-half inch within and above apex beat. Nine ounces of pus removed. No irrigation; tube drain. First improvement; two weeks later aggravation of condition in spite of good drainage.

[40] Ann. Surg., 1915, lxii, 660.

[41] Ibid., lxiii, 660.

Incision was now made in the eighth interspace at the post-axillary line, 16 ounces of pus were removed, and a tube was inserted. Resection of a portion of three ribs was done later. The patient died within a week after the last operation. In the light of post-mortem knowledge it appears that this case was a primary mediastinopericarditis, which within two months was followed by a purulent pleurisy. It would seem that the child presented a primary inflammation of the entire anterior mediastinum, which included the pericardium; the empyema was concomitant through continuity. (Edwards, W. A.[42])

Patient was a soldier with left-sided pleurisy following influenza. Empyema. Incision in sixth intercostal space; demonstration of general pleurisy, with considerable retraction of the lung. General improvement followed, but dyspnea persisted and became greatly aggravated three days after the operation. Paracentesis. Pericardiotomy. Incision 6 cm. in fourth intercostal space; escape of only a few drops of turbid fluid. Patient succumbed to asphyxia a few minutes later; the operation having been too long delayed.

Autopsy findings showed that anterior wall of heart was adherent, about a pint of pus was found behind and at the sides of the adherent heart. (Delorme and Mignon.[43])

Boy, aged seven years. Pericarditis, followed by empyema. Pericardium was reached through left fourth interspace, and a considerable quantity of seropurulent fluid. Drainage tube. Considerable improvement. The fluid removed was albuminous and coagulable, and contained Staphylococcus pyogenes aureus. Empyema subsequently developed, fluid aspirated contained the Staphylococcus pyogenes aureus. Thoracotomy yielded 30 ounces of pus. Good recovery. (Peters, G. A.[44])

Male, aged thirty-six years. Old purulent encysted pleurisy, no acute course. Resection of two inches of fifth cartilage; pleura not opened; incision of pericardium; escape of 15 to 16 ounces of pus. Irrigation with hot water; edges of pericardium were sewed to skin.

Death from weakness in fourteen hours. According to the post-mortem findings the origin of the pericardium appeared to be a collection of pus lying close to the right of the sac between it and the lung and the chest wall, which was the result of a previous pleurisy. (Ogle, C., and Allingham, H.[45])

Boy, aged eleven years. Old empyema. Resection of the sixth rib below and inside nipple. Evacuation of 2 quarts of pus. Irrigation with sterile water, gauze drain. Death in twenty days. Allen; quoted by Roberts.[46])

[42] Transactions of the Medical Society of the State of California, 1893, p. 166.
[43] Rev. de Chir., 1895, xv, 1008.
[44] Edinburgh Med. Jour., 1903, xiii, 209. Purulent pericarditis complicated by empyema; operation and recovery.
[45] Lancet, March 10, 1900, i, 693. [46] Am. Jour. Med. Sc., 1897, cxiv, 662.

ACUTE PERICARDITIS.

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This paper is based upon a study of the protocols of eighty proved cases of acute pericarditis and the literature particularly of the last five years. It seeks to emphasize certain physical signs which have seemed of importance to the writer.

Most authors, especially the writers of text-books, begin with a history of pericardial lesions. There is no record that Hippocrates observed the condition, but Galen noted it in animals and suspected it in man; various writers mentioned it, but it was not referred to as a clinical condition until described by Senac in 1749. Laennec, the father of auscultation, described a creaking sound over the heart, but it remained for Collin, his chief assistant, to clearly demonstrate the pericardial friction rub and interpret its clinical significance.

Anatomy. It is important for the student of pericardial inflammations to rehearse the anatomy of the pericardium—to have the anatomical relations of the sac to other structures in the thorax in mind is helpful in considering the subjective and objective symptomatology. The pericardium is a conical serofibrous sac enveloping the heart and the first portion of the great vessels. The apex is directed upward and surrounds the great vessels about two inches above their origin from the base of the heart. Its base is attached to the central tendon, and part of the adjoining muscular structure of the diaphragm extends a little farther to the left than to the right side. It is separated from the sternum in front by the remains of the thymus gland above and a little loose areolar tissue below, and is covered by the margins of the lungs, especially the left. Posteriorly it forms the anterior boundary of the posterior mediastinum resting upon the descending aorta, the bronchi, esophagus, trachea, and root of the left lung. Laterally it is covered by the pleuræ, the phrenic nerve, with its accompanying vessels, descending between the two membranes on either side. There are two layers, a strong dense fibrous layer and the serous layer, which invests the surface of the heart—Cohnheim[1] observed that normally the parietal layer is always in immediate contact with the visceral.

Considering the intimate contact of the pericardium with almost all the structures of the thorax, it is not remarkable that pericarditis may be manifested in almost any portion of the thoracic viscera or even the abdominal, but still more remarkable that it may exist without giving a sign—Cohnheim pointed out that complete oblitera-

[1] Lectures in Pathology.

tion of the sac was often enough a postmortem discovery for which no symptoms during life had prepared us, and Osler confesses that it is more often discovered in the autopsy room than in the ward. In some cases this may be accounted for by lack of watchfulness on the part of the clinician, by the failure to correctly interpret the relationship of various physical signs, a too common fault, or the lack of attention to the heart when pericarditis comes late in a progressively fatal case.

Etiology. The principal change in recent years in the etiology of pericarditis is to regard all cases as of infectious origin. Osler,[2] in 1902, said that so-called idiopathic pericarditis was rare, but cases could occur, chiefly in children, in whom there was no evidence of rheumatism or any local or general disease. McFarland[3] says that there is always a focus of infection, but it is sometimes undiscoverable, and he quotes Bauer, of Munich, who states that out of 3000 autopsies occurring yearly in his city he sees only two or three cases of so-called idiopathic pericarditis. Demiéville[4] found but one case in the literature of twenty years, Virchow's case of hemorrhagic tuberculous pericarditis. Mallory[5] says that a variety of infectious agents may cause acute inflammation of the pericardial cavity. The most common are the Diplococcus pneumoniæ, the Streptococcus pyogenes, the Staphylococcus aureus, and the tubercle bacillus. They probably never lodge here primarily, but come from lesions elsewhere—thus by direct extension or through lymphatics from the lung, mediastinum, or heart, or by the blood stream from any part of the body.

Preble[6] in his series found pericarditis as a complication in pneumonia, 34 per cent; rheumatism, 28.36 per cent.; chronic diffuse nephritis, 11.20 per cent.; tuberculosis, 10 per cent.; sepsis, 4.7 per cent.; aneurysm, 2.6 per cent.; typhoid, 1.7 per cent.

Sears[7] in his report found rheumatism associated in 51 of his 100 cases, and a previous history with 40 others. Pneumonia or infection with the pneumococcus was next with 18. In 7 cases chronic nephritis was the chief cause and in 5 pleurisy—chronic rheumatism and gonorrhea are both credited with 2 cases while 1 occurred in each of the following diseases: empyema, tonsillar abscess, acute nephritis, hepatic cirrhosis, and chronic fibrous pneumonia, which was apparently not tuberculous. There were 9 cases of idiopathic pericarditis, but when we consider that some dated back as far as 1882 it is not remarkable that 9 out of 100 should be so classed.

Lamar and Meltzer[8] produced pneumonia in forty-two dogs by

[2] Practice of Medicine, 1902.
[3] Jour. Am. Med. Assn., December 7, 1901.
[4] Rev. méd. de la Suisse romande, Geneva, 1911, xxxi, 449.
[5] Principles of Pathological Histology, 1914.
[6] Jour. Am. Med. Assn., December 7, 1901.
[7] Med. and Surg. Reports, Boston City Hospital, 1897.
[8] Jour. Exper. Med., 1912, xv, No. 2.

means of intrabronchial insufflation, using pure cultures of pneumococci in suspension. They produced a lobar pneumonia in each instance. The findings in the fatal cases were similar to those in man: empyema, pericarditis, septicemia. In the fatal cases the pleural cavities contained a large quantity of bloody fibrinopurulent exudate, the pericardium was inflamed, and a septicemia existed. The exudate from the lung, pleural and pericardial cavities contained as well as the blood, large numbers of capsulated pneumococci in pairs and short chains. In their non-fatal cases it does not appear that the pericardium was involved, although much the same lung area was consolidated, but not as extensively. It would appear, therefore, that these results indicate that the pericarditis was the result of a profound septicemia.

The writer, working in the laboratory of Professor H. T. Karsner, produced pneumonia in ten dogs by the intrabronchial insufflation of pneumococcus cultures. Every effort was made to prove the organism before and after injection. One dog died on the tenth day, showing inflammatory changes in the lungs, kidney, spleen, liver, and heart. In the other nine dogs the pneumonia ran a mild course, the kidneys and liver showed moderate changes, but the hearts were normal. As far as the heart was concerned in the writer's fatal and non-fatal cases the involvement occurred only in the presence of a profound septicemia.

Torrey and Rahe[9] in their studies in canine distemper occurring in a series of ninety dogs found areas of consolidation in over 90 per cent. of lungs, while pericarditis was observed in only 5 cases, all terminating fatally. In 2 the condition was associated with pleuritis, and in 1 instance, Streptococcus pyogenes, while in the others it was sterile. The Bacillus bronchisepticus readily produces pneumonia but not pericarditis.

E. C. Rosenow[10] shows in his animal experiments with streptococci from rheumatic fever the greatest number of cases of pericardial infection corresponding with the observations of various clinicians. The interesting clinical point is that when he allows a reduction in virulence by keeping the organism in culture media there is a marked falling off in the effect of the organism on joints, myocardium and endocardium, while the pericardium does not become involved; but when the virulence is increased by animal passage the effect is less upon the joints and more on the endocardium and pericardium, especially the pericardium.

Robin and Fiessinger[11] state that gonococcus infections seldom attack the pericardium. These authors quote d'Argis, who, in 1895, collected 13 cases, and they report one case of their own. Their patient was a male, aged twenty-five years, with urethral discharge

[9] Jour. Exper. Med., January, 1913.
[10] Jour. Am. Med. Assn., November 13, 1915.
[11] Soc. méd. des. hôp. de Paris, 1912, xxxiv, 802.

and acute rheumatism complicated by signs of pericardial fluid. They aspirated at Marfan's point (base of xiphoid) and removed a fibrinous serum which did not show gonococci. The patient was treated with injections of Wright's vaccines, and made a good recovery.

The writer has not seen a case of gonorrheal pericarditis, and in the 78 Boston City Hospital autopsy cases in the last ten years there is not one recorded.

Triboulet and Harvier[12] report a case of severe typhoid in a boy, aged eleven and a half years, death being due to perforation. Acute pericarditis was unsuspected during life, but at the autopsy 90 grams of fluid were found in the pericardial sac. Cultures from the fluid gave a growth of the typhoid bacillus.

A. Pissary[13] mentions pneumonia, bronchopneumonia (especially in children), gonorrhea, scarlatina, typhoid, tuberculosis, puerperal septicemia, streptococcus infections, but places rheumatism first in etiology.

Mobécourt[14] gives an interesting case of streptococcus pericarditis in an infant; infection through the umbilical vessels at the time of birth produced a purulent peritonitis, with secondary purulent pericarditis. The purulent form, this writer thinks, is frequent in infancy because there is small defence against pyogenic organisms. This has not been the experience of Barkan and Lucas,[15] but all these authors (Bovaird[16]) agree that in small children the pneumococcus is the most frequent cause. Their articles contain exhaustive statistics on the etiology in infants and small children.

Marfan[17] says that pneumonia in children under six years and in older children and adults, acute articular rheumatism are the primary causes of pericarditis.

Of the 78 proved cases in our series, in 58 some form of acute infection was recognized before death. Pneumonia was diagnosed 28 times and acute pneumonia or pleuritis was found 49 times at autopsy. Of the 28 diagnoses of acute pneumonia, acute pericarditis was discovered before death in only 4 instances. It occurred in diphtheria 4 times, in chronic interstitial nephritis, 3; appendix abscess, 1; cerebrospinal meningitis, 2; scarlet fever, 5; general peritonitis, 3; chronic cardiac, 4; malignant endocarditis, 3; gallstones, 1; thoracic aneurysm, 3; erysipelas, arteriosclerosis, ulcerative colitis (TB) and gangrene once each. The clinical diagnosis of acute arthritis does not appear in the series, and while we have all seen acute pericarditis accompany this condition many times the mortality must be low as compared with pneumonia.

[12] Bull. Soc. méd. de hôp. de Paris, 1911.
[13] Clinique, Paris, 1913, viii, 756–759.
[14] Jour. de méd. de Paris, 1913, xxv, 383–388.
[15] Boston Med. and Surg. Jour., 1912, clxvi, 444–448.
[16] Med. and Surg. Report, Presbyterian Hospital, New York, 1912.
[17] Semaine méd., Paris, 1913, xxxiii.

The question of extension of the inflammation from adjacent organs, especially in pleuritis and pneumonia, seems still in doubt. Osler[18] believed that it could occur by extension as a serious complication in pneumonia, and he reported, in 1902, that it was present in 5 cases of 100 postmortems he made at the Montreal General Hospital. He states that in simple pleurisy it is rare.

G. A. Gibson,[19] in 1898, said that it often had its origin by extension in pleurisy and pleuropneumonia as well as in endocarditis and myocarditis, but he furthermore states that it must not be forgotten that in pneumonia the source of the pericardial infection may be through organisms conveyed by the blood, and pericarditis from pneumococci has been seen without pneumonia. Septic diseases of the mediastinal glands, mediastinal tumor (Bergmann[20]), as well as destructive processes in the lungs, may produce pericarditis by extension, while acute affections of the bones—the sternum and ribs in front and the vertebræ behind have been seen to give rise to it. In one of our cases it was due to sarcoma of the mediastinum, but it was not classed as acute pericarditis.

Preble[21] lays down rather definite statistics showing the ratio of pericarditis to the area of lung involved in pneumonia. He says that with unilobar pneumonia the chances of a pericarditis are 1 in 40; with a bilobar or trilobar, 1 in 10; with a quadrilobar, 1 in 5; with a pneumonia of the entire left lobe, 1 in 8. He feels that the danger is somewhat greater with a left than with a right-sided pneumonia. The writer has not found another author who has laid down such definite lines.

Mallory in his book states that pericarditis may occur by direct extension, and Professor Councilman makes a similar assertion in his lectures.

Sears[22] in a study of 100 cases found 34, with evidence of some previous valvular or myocardial affection, and while he says it may be a coincidence, the number is so large that it is more probable that the preëxistent lesions acted as a determining cause. Many of his cases were complicated by, or secondary to, pneumonia, pleurisy, or both, either by direct extension or from a simultaneous infection.

Brooks and Lippencott[23] found in their 150 cases of pericarditis, pleural lesions present in 136. In well over half of these cases it appears that the pleural change is the more anterior, and that the pericardial alteration occurred later, though still back of this relationship is the general disease or condition in the course of which the two membranous inflammations developed. They found a direct relationship between pericardial and pleural lesions best

[18] Practice of Medicine, 1902.
[19] Diseases of the Heart, 1898.
[20] Charite-Ann., Berlin, 1909, xxxiii, 92–101.
[21] Loc. cit.
[22] Loc. cit.
[23] Am. Jour. Med. Sc., December, 1909.

shown in the acute serofibrinous cases, in which out of a total of 67 instances, 60 showed pleural changes. In 36 of these the lesions were identical. These authors were unable to trace any direct relationship between pericardial and endocardial disease. For example, in their serofibrinous cases, 46 of which were caused by bacteremia of some variety, only 10 showed acute endocardial changes; 7 of these were unquestionably rheumatic and bore no other relation to the pericarditis than that both had probably a common etiology.

Sinnhuber,[24] believes that acute articular rheumatism is the foremost cause of pericarditis, then pneumonia, tuberculosis, scarlet fever, influenza, typhus abdominalis, recurring fever, cerebrospinal meningitis, malaria, erysipelas, osteomyelitis, pyemia and sepsis, gonorrhea, scurvy, leukemia, and certain forms of cirrhosis of the liver. He also states that it can arise from diseases of the neighboring organs, endocarditis, myocarditis, diseases of the pleura, especially on the left side, perforation of the esophagus from a foreign body, caries of the sternum and ribs, and as a sequel of aneurysm.

In our series cultures were not always made, but in the 78 cases the streptococcus was recovered 19 times, the pneumococcus 17 times and staphylococcus 8. In one of the cases of cerebrospinal meningitis the meningococcus was recovered from the brain and pericardium, and in the other case the heart's blood showed streptococci, and the pericardium, staphylococci.

Symptoms. The symptoms of pericarditis are so varied and the onset so insidious in some cases that Osler and almost every other author has been led to say that many cases run their entire course without recognition. When it comes on very gradually, or is the only manifestation of an obscure focus, or is well established when the patient comes under observation, detection is sometimes most difficult if not impossible. The importance then of a thorough physical examination of the heart and lungs at the first visit, with subsequent daily examination augmented by careful notes and charts, is obvious. It is well to mark out the percussion lines on the chest and to test them daily for any change. The intensity of all the heart sounds, their relation to one another, and the character of murmurs should be carefully noted.

Pain is an important subjective sign and one which a good many practitioners expect to find. Pleurisy is almost immediately painful, and so it is thought that pericarditis will be; but one should not expect to have the patient complain of pain as he does in pleuritis.

A. McPhedran[25] says that pain is generally present, and quotes Sibson, who found it in 70 per cent. of his cases. L. V. Schrötter[26]

[24] Die Erkrankungen des Herzbeutels und ihre Behandlung, Berlin, 1911.
[25] Osler's Modern Medicine, 1908. [26] Nothnagel's Practice.

states that the greater number of cases occur without pain. James Mackenzie[27] calls attention to the absence of pain in a good many cases. Poynton[28] says it may occur with little discomfort and no pain. Pain was present in 65 cases in the series of 100 reported by Sears, and when absent its place was taken by a sense of oppression in the chest.

Babcock[29] found pain in the majority of his cases, or if not, a sense of distress.

It has seemed to the writer that a good many patients with acute disease of the heart, while they do not have actual pain in the heart, still when one asks if ice over the precordia makes them feel more comfortable, they generally say that they prefer to have the ice kept on.

Blumer[30] states that pericarditis is often almost without pain.

Billings[31] does not mention pain in his text, and in only one case history.

In the protocols of the 78 cases in our series, pain was not a striking feature, and was observed 26 times—in 17 of the 26 cases there was either acute pneumonia or disease of the pleura, in 2 others chronic disease of the myocardium, and in 1 aneurysm of the arch of the aorta, leaving but 6 cases out of the 26 with pain directly over the precordia.

In the 2 cases of purulent pericarditis with operation (one recovered, the other died without autopsy) pain was not observed at any time.

It has been the writer's experience repeatedly in cases of slight dry and serous pericarditis lasting a few days to note the absence of pain, although in this study no conclusions from figures can be given.

It must be remembered that many of the patients in our series were seriously ill at the time of entrance to the hospital, and their general distress was so great that they may not have noticed pericardial pain.

The cause of pain is difficult to determine. The pericardium, like other membranes, the peritoneum and pleura, are insensitive when normal, but if inflamed, pain is produced. Special work seems necessary on this subject.

Poirier[32] says it is very probable that the left pneumogastric and the nerves of the cardiac plexus give off also several nerve fibers to the fibrous layer.

The skin over the precordium is sometimes tender because of the connection between the upper intercostal nerves and the ganglia and nerves of the cardiac plexus (Piersol[33]).

[27] Diseases of the Heart, 1908. [28] Pediatrics, New York, 1910, xxii, 353-363.
[29] Diseases of the Heart and Arterial System, 1905.
[30] Jour. Am. Med. Assn., July 18, 1914.
[31] Ibid., December 7, 1901.
[32] Anatomy. [33] Anatomy.

Krehl[34] quotes Goldscheider,[35] who denies the existence of special nerves of pain. He continues with the statement that every sensation produces at the same time a more or less definite impression of the place whence the sensation has come. In the case of the eyes and skin the localization is very accurate; in the case of the mucous membranes near the outside of the body it is somewhat less accurate; in the case of the mucous membranes and the organs within the body it is inaccurate and entirely unreliable. Howell[36] also makes this last statement.

Albutt[37] states that there are cases in which the pain is like that of angina pectoris, and refers to several observers, Sibson, Byron Bramwell and others.

In studying the subjective symptoms of pain and a feeling of distress in the precordia or chest in pericarditis, it seems to me that it makes a great difference whether one builds his statistics of pain on clinical or autopsy observations. When one considers that only about 50 per cent. of the cases are recognized, and of that number about one-half have precordial pain, it makes the pain a small factor.

Pericardial Friction. In a pericarditis which remains dry or rather has but a moderate increase in fluid the sole physical sign is the to-and-fro friction rub. It is generally heard, if only for a few hours, in almost every case of plastic pericarditis.

Billings[38] says it is present in every case at some time in its course, and by systematic examination it will be found.

McPhedran[39] thinks there are many cases in which it is not found, "but once distinctly heard it can scarcely be confounded with anything else." The writer feels hardly able to make such a sweeping statement. Meeting a considerable number of students of all ages every year in the Graduate School of Medicine of Harvard University, it is uncommon to have a student recognize a pericardial friction rub. Three years ago a very able practitioner from Vermont told me that he had been in practice thirty years and had never heard a pericardial rub until he came to our clinic. This is not surprising when one considers that acute pericarditis is not such a very common complication, having been diagnosed clinically but 100 times in 34,467 medical patients treated in the wards of the Boston City Hospital during the last ten years by physicians giving a good deal of time and thought to diseases of the chest. Acute pericarditis has been found 78 times in 1553 autopsies during the same period.

Many writers speak of the pericardial changes beginning at the point where the membrane is in contact with the great vessels,

[34] Clinical Pathology, 1909.
[35] Ueber den Schmerz, Berlin, 1894.
[36] Text-book of Physiology, 1913.
[37] Diseases of the Arteries, 1915.
[38] Loc. cit.
[39] Loc. cit.

because the infecting agent is carried to the sac by the blood, and at that point the blood supply is richer.

Rosenow[40] found in the pericarditis of animals that hemorrhages usually surrounded a relatively large artery. In some of the sections the artery was the seat of thrombosis, or accumulation of leukocytes along the intima in the area of the hemorrhages. Some of the leukocytes contained organisms, and in one instance typical organisms were found directly in the wall of an artery which showed mural implantation of leukocytes adjacent to an area of hemorrhage in which exactly similar organisms were found in small numbers.

Mallory[41] says that it may occur anywhere in the pericardial sac. In one case of the series of 78 autopsies of acute pericarditis the inflammation was found about the great vessels, but more often it involved the entire sac. In animals the focus can be more readily studied while the human subject often does not die until the entire sac is involved.

In the experiments of Walker and Christian[42] in the production of pericarditis by the injection of spartein sulphate and adrenalin chloride, the inflammation was usually adjacent to the left ventricle.

The chief difficulty seems to be in distinguishing the rub from a valve murmur. The rub even when faint will generally have a jerky, grinding, or leathery quality. Because of the more active movements of systole it may seem to be wholly a systolic sound, but by listening carefully a sound of similar quality, sometimes very faint, will be heard in diastole. The rub may be mistaken for a valve murmur even by the most experienced. Three years ago, when looking for a case of aortic stenosis to show the students, I was told by the house physician of a colleague, distinguished as a specialist in circulatory diseases, that they had a case in Ward M. The patient, a man, aged twenty-seven years, was convalescent from an acute multiple arthritis. He looked well and said that he was absolutely free of his discomforts. On listening to his heart he was found to have over the second interspace to the right of the sternum a rough, grating murmur, systolic in time. On listening carefully, however, a similar murmur, very faint, was heard in diastole. There were absolutely no cardiac or circulatory signs indicating aortic stenosis or regurgitation. In the absence of these confirmatory signs of valvular disease, outside of the precordia and taking into account the history of recent rheumatism, it seemed more probable that he had a pericardial friction rub. Two days later the rough sound, with its fainter diastolic, was heard over the third interspace to the left of the sternum, the following day over the fourth, and by the fifth day it was barely audible just above the cardiac impulse, and then disappeared. Within two months a consultation was held at the hospital to decide whether a patient

[40] Jour. Infect. Dis., May 16, 1915.
[41] Loc. cit.
[42] Tr. Assn. Am. Phys., 1910.

had double aortic disease or pericarditis. The same considerations decided in favor of pericarditis.

The rub often moves from day to day, and is sometimes made more intense by pressure with the stethoscope. When the rub disappears it may mean that the plastic pericarditis is quieting down or fluid is accumulating.

Fluid. In many cases, the majority in fact, a little increase in fluid occurs and then quiets down. It is not recognized clinically, as it gives no physical signs because the sac is distensible and the added pressure causes no embarrassment to the heart or circulation.

Poynton[43] says that paracentesis is rarely needed in childhood, thereby indicating the frequency of spontaneous absorption.

The cardinal points which have always been taught are increase in the cardiohepatic angle, increasing faintness (distant) of the cardiac sounds, the disappearance of the cardiac impulse, and the pulsus paradoxus.

Percussion should be performed daily or oftener marking out carefully the cardiac borders. The angle of cardiohepatic dulness will be found to increase with increasing fluid. James Mackenzie[44] says that the dulness will also reach up to and above the second rib and the area mapped out will have a somewhat pear-shape appearance. This shape has been seen several times by the writer, and in a roentgenogram the enlarged area looks like a paper bag filled with water and set down upon a table. In comparing these roentgenograms with those from dilated hearts the line in the latter is almost always a curve inward as it approaches the liver. If the increase is due to enlarged liver it can generally be distinguished by a study of other pericardial signs of effusion, such as a faintness of heart sounds and disappearance of the cardiac impulse.

Emphysema may mask the heart sounds so that there may be poor sounds with a good pulse, as is often the case in pericardial effusion. When there is a left pleural effusion the heart may be pushed to the right and the border between the pleura and the pericardium will be lost. Aspiration of the pleural fluid should reduce the cardiohepatic angle, and if it does not, after the heart has had time to return to its normal position, pericardial fluid should be suspected (Cabot[45]).

Shifting dulness has not been helpful to the writer. The fluid is generally under such tension between the myocardium and the fibrous parietal pericardium that it is not clear how it could shift. When the fluid is posterior the heart may be pushed forward and there may be no diminution in the cardiac impulse. In a case seen in consultation the increasing cardiohepatic angle and signs of distress raised the question of fluid. A roentgenogram showed the characteristic pear-shaped enlargement; still, we hesitated to aspirate

[43] Loc. cit.
[44] Diseases of the Heart, 1914.
[45] Physical Diagnosis.

because of the very distinct cardiac impulse. A second roentgenogram was taken, showing an increase in size, and upon aspiration about 150 c.c. of bloody serum were removed, with considerable relief to the patient. In these cases almost all authors, Billings,[46] Babcock,[47] J. Mackenzie,[48] Cabot,[49] Musser,[50] Parkinson,[51] call attention to the small area of dulness in the left back just inside the angle of the scapula, described by Ewart, and produced by mechanical atelectasis of the compressed lung.

Moog[52] adds one case to a report of four others of pericardial effusion punctured in the rear. He tapped the pericardium through the fifth interspace in front and evacuated 130 c.c. of hemorrhagic fluid; two days later he evacuated 90 c.c. in the same way. The symptoms from compression of the lung soon returned, but two punctures at different points brought no fluid. Then he introduced the needle in the rear in the eighth space and obtained 600 c.c. of the effusion, the same amount the next day; and five days later 400 c.c. Air got in during the later puncture, and pneumothorax developed with some effusion, but this gradually disappeared with all the other symptoms. The patient made a good recovery.

Pauly[53] calls attention to the signs in the back.

Curschmann[54] reports 3 cases of successful paracentisis in the back at the eighth interspace.

Mackenzie[55] reports having tapped a purulent pericardial effusion from the back in mistake for an empyema, his mistake arising from not having ascertained the position of the heart's movements. He says that had the case been one of pleural effusion he would have found the heart beating to the right of the sternum, but the whole left chest was dull, so that the idea of it being a pericardial effusion did not occur to him. One of my hospital colleagues told me that he made a similar mistake two or three years ago.

R. C. Larrabee and the writer tapped a boy, aged ten years, just in front of the left midaxillary line, removing several ounces of bloody fluid. The almost immediate cessation of cardiac distress convinced us that we had put the needle into the pericardial instead of the pleural cavity.

The pulsus paradoxus, which is small and feeble during inspiration, was formerly considered as important, but it occurs in other conditions and even in the normal subject.

Musser,[56] Cabot,[57] West,[58] and many others mention it, but do not regard it as important. In one case in the writer's series it was noted.

[46] Loc. cit. [47] Loc. cit. [48] Loc. cit. [49] Loc. cit.
[50] Medical Diagnisis, 1913.
[52] Therap. Monat., Berlin, June, 1914.
[53] Rev. de méd., Paris, 1911, xxxi.
[54] Therap. Monat., Berlin, 1912, xxvi.
[55] Loc. cit.
[56] Loc. cit.
[57] Loc. cit.
[58] Lancet, London, 1910, i, 560–564.
[51] Clin. Jour., London, 1912, xl, 45.

Taussig[59] says that ordinarily the dicrotic wave in the radial sphygmogram does not vary in size with the phase of respiration. In pericarditis, however, it becomes more or less exaggerated, sometimes greatly so, during inspiration. He studied 5 cases, making this observation in all.

The writer has not had experience with the following signs:

Wynter[60] believes that absence of the abdominal respiratory movement is an indication of pericarditis. He reports 2 cases in which the remarkable stillness of the abdomen led to a diagnosis of acute abdominal lesions, pericarditis being discovered later. It is a valuable sign, he thinks, as it may precede and outlast other indications. The inflammation of the pericardium may cause reflex inhibition of abdominal movement some days before the appearance of the ordinary clinical signs. It is more apparent with a fibrinous exudate. He reports 10 other cases in which he found this sign.

When we remember that the base of the pericardial sac rests upon the diaphragm it is quite as possible to give abdominal symptoms as in the well-known instances of inflammations of the pleura and lung.

In the only cases in our series in which abdominal pain was noted there was definite disease of the abdomen.

Cobb[61] has reported a case of acute serohemorrhagic pericarditis in a boy, aged eleven years. He had never been sick until ten days before Cobb saw him, when he had an acute coryza, with the exception that he complained of a little pain at the apex of the left lung on deep inspiration. His physician found nothing but a few slight friction rubs, which disappeared within twenty-four hours. The temperature did not go above 100.5° F., and became normal at the end of the third day. Three days after apparent recovery and five days before Cobb saw him he ate heartily of apples and ice-cream, was seized with a sudden attack of abdominal pain, which made him cry out and writhe on the floor. He vomited several times in this attack; the temperature was 100° F., but nothing could be found on physical examination. He recovered in a few hours and was apparently as well as ever. Three days later he had some nausea and vomiting, and complained of abdominal pain. This was followed by a day of comfort, when he again had nausea, vomiting, epigastric pain, and pain in the right hypochondrium well down in the flank. He was apparently very sick, with a rapid and irregular pulse. In the epigastrium and right and left hypochondrium there was exquisite tenderness. After careful examination of the chest, nothing abnormal was found except the respiration in the left back was somewhat shallow, apparently due to the tenseness in which

[59] Jour. Am. Med. Assn., June 13, 1914.
[60] Clin. Jour., London, 1911–12, xxxix, 218.
[61] Ann. Surg., October, 1912, lxi.

the abdominal muscles were held because of the pain. The heart was normal in size and situation. Upon opening the abdomen in the median line nothing was found except a very large and congested liver. At the autopsy the pericardium was found to be 5 mm. thick and contained a considerable amount of bloody fluid with soft fibrin, free and attached. Cultures were not reported.

The writer thinks that the shallow breathing in the left back may have been due to the pericardial pressure—Ewart's sign; the congested liver, to pressure about the great veins within the sac. The heart valves were normal.

I was once asked to see a physician's wife who had been taken to a private hospital with a diagnosis of appendicitis. Certain features in the case prompted the surgeon to ask for a medical consultation. The history was that she became easily tired and a little short of breath on exertion. She had had three attacks of abdominal pain, which seemed to bear a relation to her menstruation. On physical examination a marked mitral stenosis was found and an enlarged and tender liver. By resting the heart the liver reduced in size and the attacks of pain ceased.

Calvert[62] believes that in pericarditis with effusion the right lobe of the liver is low, varying in its position from the 5th space to the 6th rib; in dilatation of the heart the right lobe of the liver is high from the 4th to the 5th rib. He reports 2 cases of pericarditis to confirm this.

The writer has not met with this sign nor has he found mention of it elsewhere, but it seems extremely doubtful if it would give much help. Turrettini[63] studied a sign noted by Josserand,[64] and Mouriquand and Roubier[65] of impending pericarditis or endocarditis. Turrettini observed this sign, "Éclat clangoreaux disastolique au foyer pulmonaire" in 2 cases, a sudden and violent clanging diastolic sound in the pulmonary area. The pulmonary second sound was very pronounced and the vibration could be felt by the hand. The second aortic sound was perceptible, but less marked, a friction rub developed soon after. His cases are not very helpful, as one patient had chronic nephritis with hypertrophy and dilatation and the fibrinous pericarditis developed slowly while the patient was under treatment, and the other was similar without autopsy. In two other cases of pericarditis from the spread of a pleural process this peculiar clang was never heard. He believes the sound to be important because it enables one to recognize incipient pericarditis, and by instituting treatment in time to possibly ward it off. The writer does not find in the literature any other observers who have made use of this sign. In chronic nephritis the diastolic

[62] Arch. Int. Med., 1909, iii, 92; Jour. Am. Med. Assn., August 27, 1910.
[63] Rev. méd. de la Suisse romande, Geneva, 1913, xxxiii.
[64] Lyon méd., November 17, 1907.
[65] Ibid., September 1, 1907.

second sound is often sharp and ringing, until the systolic blood-pressure falls and cardiac compensation fails.

The knee-chest position has been considered by Rieux[66] to be of diagnostic value. The writer has never seen a case of pericarditis with this sign. In diseases of the chest patients assume the most comfortable attitude for breathing.

The diagnosis in dry pericarditis rests solely upon the to-and-fro friction rub. The fluid which usually follows may be so slightly in excess of the normal as to escape detection and with the disappearance of the rub nothing further may be found, or there may be a gradual or rapid change in the area, the obliteration of the cardio-hepatic angle, dulness extending to and above the second rib; the disappearance of the cardiac impulse; the faintness of the cardiac sounds; increasing cardiac distress sometimes out of proportion to the cardiac findings; cyanosis and signs of embarrassed circulation. In a man, aged thirty-two years, who had been ill two weeks with pneumonia, was getting better and then began to grow worse, the right border of the heart was 7.5 cm. from midsternum, the left border 14 cm. The apex beat was indistinctly felt in the 5th space within the nipple line. The cardiohepatic angle was distinctly obtuse. The striking point was the difficulty of hearing anything in the way of heart sounds. The abdomen was practically negative, and there was no sign (Ewart) in the back. Dr. G. G. Sears, who saw this patient shortly after he entered the hospital, immediately made a diagnosis of pericarditis with effusion, based on the rapidity of respiration, hoarseness of voice, size of cardiac dulness, and lack of heart sound with a fairly good pulse. The roentgen-ray confirmed the diagnosis. A needle inserted in the fifth space at the left outer border of cardiac dulness gave 20 c.c. of seropurulent fluid. At operation about 2 quarts of pus were removed. The patient seemed relieved, but died a few hours later.

Sears[67] has said that unresolved pneumonia is a pathological myth. If the temperature keeps up and the signs persist after due course of time, something is amiss. If we can exclude complications outside the lungs as a cause of fever, *e. g.*, otitis media, pericarditis or endocarditis, one arrives at a diagnosis by exclusion of either pulmonary abscess or empyema, and of these the latter is vastly more common. These words might also be used in discussing pericarditis; sometimes the pleural cavity is aspirated first in doubtful cases (Ortner[68]); Harrigan[69] thus excluding paracentesis of the pericardium or proving its necessity.

We will speak of paracentesis as a diagnostic as well as a therapeutic procedure. It is necessary in cases progressing unfavorably

[66] Paris méd., 1912–13, xi, 185–191.
[67] Boston Med. and Surg. Jour., November 25, 1915.
[68] Deutsch. med. Wchnschr., Leipzig., 1910, xxxvi.
[69] Ann. Surg., lvii, 1913.

to determine the plan of treatment. The point of puncture is decided by the physical signs and the roentgen-ray.

Pendlebury[70] says the spot chosen for introduction of the aspirating needle, of not less than one-tenth in diameter, is in the 5th left interspace, one inch from the margin of the sternum. By choosing this spot injury to the pleura and danger of wounding the internal mammary artery will be avoided. The most careful aseptic precautions must be observed, and he advises incising the skin before putting in the needle. He urges careful attention to the first few drops of fluid to avoid injury to the heart. These directions are also given by Friedrich Pels-Leusden,[71] who adds that we may in the above method occasionally prick the heart, and furthermore says that we may go in the mammary line in the 5th space in which the mammary artery and the heart are not imperilled, but where we must necessarily go through the pleura. Curschmann advises puncture always far outside at the limit of dulness and to go without fear through the pleural cavity, even if the latter is matted down by adhesions, so as to be sure not to injure the heart.

Ogle[72] advises introduction of the needle slowly and steadily, and as soon as it is through the skin the aspirator should be connected and the glass index watched for the first indication of fluid.

The writer has had his chief experience with the above method. Blechmann[73] in an exhaustive article discusses the various methods of exploratory puncture. The one already described by Pendlebury (Dieulafoy[74]), the left parasternal (Delorme and Mignon[75]), the right parasternal (Rotch[76]), and Marfan's[77] epigastric xiphoid route.

As Curschmann says, no definite rule can be laid down in any case. We must be guided by the area of dulness, the findings by exploratory puncture and the roentgen-ray.

In our other case of purulent pericarditis following pneumonia the right border of cardiac dulness extended until within 2 cm. of the right nipple, and exploratory puncture was made in the 4th space to the right of the sternal margin and the operation followed the same line, the 4th and 5th costal cartilages being resected. This patient made a good recovery and the discharge ceased in three weeks.

The chief worry of the novice in exploratory puncture is the fear of wounding the heart. Sears[78] says: "That the danger arising from wounding the heart is theoretical rather than real received

[70] Latham and English, System of Treatment, 1915.
[71] Handbook of Surgical Operations.
[72] Latham and English, 1915.
[73] Semaine méd., 1913, xxxiii.
[74] Traité de l'aspir. des liquides morbides, Paris, 1873.
[75] Rev. de Chir., 1895, 797–787.
[76] Boston Med. and Surg. Jour., 1878, xcix.
[77] Semaine méd., Paris, 1913, xxxiii, 469–476.
[78] Boston City Hospital, Med. and Surg. Report.

another illustration in one of these cases when the needle thrust into the 6th interspace just to the right of the sternum entered a solid body which pulsated and caused it to describe an arc of one and a quarter inches with each beat. No harm results."

Ewart considers the removal of blood sometimes most beneficial, and Sharkey[79] reported a case in which the blood came through the trocar in jets. It did the boy a great amount of good.

Byron Bramwell[80] reports a case of continued hemorrhage into the pericardium from puncture of the ventricle.

In rabbits, blood is removed from the ventricle for therapeutic purposes without doing the rabbit any harm.

Cushing and Branch[81] in their report on work done in the production of chronic valvular lesions in dogs found that in spite of the most rigid asepsis neither the pericardium nor the pleura withstand operative measures with anything like the resistance which the peritoneum exhibits in the face of similar manipulations. A considerable number of the animals which recovered and were subsequently sacrificed showed evidences of a slight degree of infection. They add, however, that no one who has not had experience with operations on animals can have any idea of the amount of handling the heart will stand without injury.

In Harrigan's[82] case of temporary arrest of the heart beats following incision of the pericardium for suppurative pericarditis, the heart was seen lying still, but it began to beat again as soon as gauze was introduced into the sac. This was a feeble child much exhausted by a long-standing infection, but recovered from the immediate effects of the operation.

Sears calls attention to the fact that the heart is more apt to show signs of embarrassment with arrhythmia and enfeeblement of the pulse when the effusion begins to form, later when the sac begins to distend the circulatory signs are less urgent. In accord with this are the findings of Heitler,[83] who electrically and mechanically stimulated the visceral pericardium, thereby considerably disturbing the cardiac rhythm even after section of the cardiac nerves. He found the left ventricle excitable, especially over the upper third; the right ventricle is not markedly excitable except close to the sulcus, while the apex is variable.

Mackenzie[84] has never found any serious embarrassment from extensive pericardial effusion. The pericardium normally is an inelastic bag, but with inflammation it becomes distensible.

Chatin[85] found that the amount of fluid which could be forced into a normal pericardium was 700 c.c., but in inflammation very

[79] Lancet, December 6, 1902.
[80] Clinical Studies.
[81] Jour. Med. Research, February, 1908.
[82] Loc. cit.
[83] Wiener klin. Wchnschr., 1898, No. 3.
[84] Loc. cit.
[85] Rev. de méd., June 10, 1900.

large amounts may accumulate. Kay[86] reports a case in which at autopsy 3500 to 4000 c.c. were found.

When the fluid is in large amount the pressure symptoms on the auricles and great veins may become marked, and death may result. Another danger (M'Phedran[87]) is the interference by pressure with the coronary blood supply, this favoring degenerative changes in the structure of the heart.

TREATMENT. The question of injection into the pericardial sac after removal of the fluid is unsettled. Few observers can see any advantage, and almost all regard attempts at local medication to be harmful. Washing out the sac even with normal salt solution is without supporters. The object of any form of local treatment is to prevent adherent pericardium.

Wenckeback[88] obtained good results by introduction of air. He removed the fluid and put in one-half the volume of air. He suggests using nitrogen or oxygen.

Wilbur[89] quotes McPhedran, who advises in pyopericardium the injection of a 2 per cent. liquor formaldehyde in glycerin, as is sometimes used in empyema.

Rehn[90] injected dogs' hearts with tincture of iodin and with aleuronat, produced synechia, and then checked adhesions by injections of sesame oil. Wilbur suggests the use of some oil, such as liquid petroleum.

In pyopericardium, free incision with drainage is indicated as in empyema. Manual removal of fibrin is sometimes necessary.

Medical treatment is thoroughly discussed in several good text-books. Ice is of value and is used in every case in the Boston City Hospital whether there is pain or not. I always ask the patient if it gives him comfort, and he generally replies in the affirmative. Be sure that the ice-bag is not too heavy—it is sometimes an added burden to a laboring chest.

Rubino[91] found that he was able to produce pericarditis in animals by the intravenous injection of cultures of pyogenic cocci only when ice was simultaneously applied to the chest. On this ground, ice has been objected to as a therapeutic measure, but Rubino probably severely chilled the animals and lowered their resistance.

The treatment by drugs is nearly useless, but whatever is done is generally indicated by the infective process.

In rheumatism the salicylates are of undoubted value. It has been said that they sometimes depress the heart, but it is more probable that the depression comes from the toxemia.

[86] Penna. Med. Jour., 1911–12, xv.
[87] Loc. cit.
[88] Ztschr. f. klin. Med., Berlin, 1910, lxxi.
[89] Jour. Am. Med. Assn., July 25, 1914.
[90] Arch. f. klin. Surg., Berlin, 1913, cii.
[91] Arch. Ital. de Biol., 1892.

Pericarditis occurring before the crisis of pneumonia is apt to mean an overwhelming toxemia, and the prognosis is very grave. Later, after the patient has begun to improve, the effusion is often purulent, and the treatment has been discussed.

The diet should be light and easily assimilable, as in cardiac and acute infections.

Absolute rest in bed until the signs of pericarditis have ceased and the heart has fully regained its tone is of vital importance.

Conclusions. The conclusions from the literature and the series of cases are:

1. Acute pericarditis is a secondary affection. Several other lesions were found in every case in the 78 autopsies.

2. The extension from adjacent structures is probably uncommon, the vast majority of cases occurring from infection of the sac through the blood stream.

3. Pain in the precordial area is not as common as is supposed. A good many mild cases pass unnoticed because there is neither pain nor distress. In rheumatism with an acute heart the pain and arrhythmia may be noticeable at first, but later when all structures of the heart are involved the picture is one of general cardiac and circulatory embarrassment. Pain is so much more common in pleuritis, and pneumonia plays such a factor in pericarditis, that it is often difficult to separate the two.

4. The difficulty in diagnosis is illustrated by the fact that acute pericarditis was recognized clinically only 100 times in 34,467 cases and 12 times in 78 autopsies at the Boston City Hospital. Pneumonia is the chief agent in the production of purulent and of fatal pericarditis. Rheumatism may cause a greater number of cases of all types, but the figures are open to doubt, as they include all grades of severity, and many can be studied from their inception, while in a rapidly progressing infection like pneumonia the changes in the heart may be masked by adjacent physical signs and the cardiac and respiratory distress of the patient. Rheumatic pericarditis should always be thought of, especially in young adults, when the signs of cardiac failure are out of proportion to the other cardiac physical findings. Pneumococcus pericarditis or myocarditis, or both, should be considered, especially in young or middle-aged adults, when the heart shows failure of compensation before the crisis or after it when there is fever and delayed convalescence.

5. The prognosis in acute pericarditis following acute arthritis is generally favorable to life; the extent of damage to the subsequent function of the heart eannot be determined, of course, immediately. In the clinical protocols of this series, acute arthritis was not found once. Pneumococcus pericarditis is grave at any stage. Tuberculous pericarditis is not common, and is usually a late involvement in an advanced case.

6. In mild cases of fibrinous pericarditis or of moderate effusion,

especially when an accompaniment of acute arthritis, treatment of the infection and local applications to the precordia will often suffice. When the progress is unsatisfactory, the physical signs increasing, and the diagnosis doubtful, paracentesis is imperative.

7. Frequent examinations during the infection and for some time after are very important. A thorough search for and the eradication of foci should be conducted in every case.

The writer wishes to thank Dr. Sears, Dr. Councilman, and Dr. Mallory for many valuable suggestions, and the members of the Visiting Staff of the Boston City Hospital for the privilege of referring to the records of their cases.

A TREATMENT OF GASTRIC ULCER BASED UPON ESTABLISHED CLINICAL, HISTOPATHOLOGICAL AND PHYSIOLOGICAL FACTS.[1]

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Even a casual acquaintance with the literature of peptic ulcer demonstrates that treatment of the affection has been largely empirical. Various types of therapy appear to have obtained a vogue because their application either relieved symptoms or appeared to prevent a fatal issue. Success of a mode of treatment has been judged mainly from its effect upon the immediate condition and not with respect to the ultimate outcome of the affection. It is most unusual to observe statistics of hospitals or private practices that indicate the status of patients treated after intervals of five to twenty-five years. Hospital records generally state that a peptic ulcer patient has been discharged either "cured," "improved," "not improved," or has "died." With exception of the fatal cases, facts are usually not available regarding the future course of the ailment, inasmuch as these patients frequently seek the advice of another physician should their disability return after a long and expensive course of treatment at the hands of a former physician. It is also of interest to observe that of a half-dozen experts nonsurgically treating peptic ulcer by radically different regimes the percentages of cure show a range of but five to ten points. We have also been impressed by the fact that at the most competent hands, certain ulcers resisted all types of treatment alike. It has seemed to us that unsatisfactory as the treatment of peptic ulcer might be,

[1] Read at the Annual Meeting of the American Gastro-enterological Association, Washington, D. C., May 8 and 9, 1916.

it were possible to devise a type of management that at least had the merit of resting upon certain basic principles. It likewise appears to us that methods of therapy are dependent upon information that has been returned by recent histological, physiological, and clinical investigations.

I. The Problem.

1. *Clinical.* Peptic ulcer is a disease which may or may not exhibit symptoms or signs. It is an ailment which, in its uncomplicated form, may be so closely mimicked clinically by nonulcerous dyspepsias as to deceive the most experienced diagnostician. Until complications occur it is a disease characterized in 84 per cent. of instances by a peculiar "periodicity," and which often manifests itself by recovery and relapse frequently wholly independent of type or duration of treatment. It is associated with a form of gastric malfunction whose cessation does not generally indicate eradication of its exciting causes. It is a disease about which little prophecy respecting its extent or its future course can be made from its clinical symptomatology. Acuteness of symptoms does not wholly indicate the histopathological changes existing at the diseased focus—for acute manifestations histologically can be engrafted upon chronic inflammatory tissue pathologically. The most serious clinical outcome may frequently follow upon spontaneous or so-called therapeutic cure with the subsidence of the initial dyspeptic upset. Stenoses, gastric malformation, involvement of extragastric viscera, fatal hemorrhage, or malignant transformation may occur with comparatively slight clinical manifestation or certainly with relatively little change in the patient's complaints. Peptic ulcer is an ailment entirely indistinguishable clinically from the early appearance of gastric cancer, syphilis, or tuberculosis. It is a disease which but rarely occurs in individuals not already affected with other clinical abnormalities—infections about the head, throat, and neck; abdominal disease, *i. e.*, inflammatory changes in the appendix, gall-bladder, perigastric lymph nodes, pelvic structures, etc.; disturbances in the circulatory, eliminative, nervous, or lymphatic systems; and malfunctions of that group of glands concerned with food digestion and assimilation. Finally, it is an ailment of almost universal distribution, and one capable of producing such disturbance of the physical and mental health as to demand relief.

2. *Nature of Peptic Ulcer as Shown by Experimentation.* Before discussing the possibility of curing an ailment, it would appear quite essential that one be adequately informed regarding the character of the existing malfunction. That such inquiry is especially warranted with respect, gastric ulcer is indicated by the observation that scarcely a year has passed since the classic monograph of Virchow that has not seen a new attempt at explaining

the cause of ulcer of the stomach. Usually, closely attached to this experimental or clinical investigation there has also been cited a supposedly new method of healing the affection. That the majority of these contributions have proved unsatisfactory is clearly indicated by a recent elaboration of the "corrosion theory" of the origin of peptic ulcer, a theory pronounced inadequate forty years ago by von Leube, and often since revived.

Study of the affection demonstrates that a narrow conception of gastric ulcer is impossible. Competent observers have experimentally produced gastric ulcer by a wide variety of methods. It has been shown that gastric ulcers differing slightly in type histologically can be caused equally as readily from the systemic as from the local intragastric point of attack. Some of the more familiar causative agents are: (*a*) *Bacteria* (pneumonococci by Dieulafoy; staphylococci by Widal and Meslay; Staphylococcus aureus by Letulle; B. pyocyaneus by Charrin and Ruffer; B. dysenterii by Chantemesse and Widal; lactic acid bacillus and B. coli by Rodet and Zaidmann; streptococcus by Rosenow); (*b*) *Bacterial toxins* (pyemias by Lebert and by Cohn; diphtheria toxin by Enriguez and Hallion and Rosenau and Anderson); (*c*) *cutaneous burns* (Welty, Ponfick, Silberman, Parascandolo); (*d*) *poisons of metabolic origin* (Bolton's "gastrotoxin" injected intraperitoneally or subcutaneously); (*e*) *extrinsic poisons* (mercury salts, arsenious acid, cantharidine, vegetable alkaloids, pilocarpin and atropin, copper sulphate); (*f*) *poisons introduced into the stomach* (corrosives, caustics); (*g*) *alterations in the stomach's circulation* (vascular blocking, adrenalectomy, thrombi, emboli, arteriosclerosis or nervous inhibition, external pressure); (*h*) *artificial pyloric spasm or stenoses* (Hamburger and Friedman); (*i*) mechanical trauma. Whatever may be the experimental mode of production, the types of ulcer resulting appear to vary but slightly. The lesions produced are essentially similar. There is inflammatory edema, loss of surface epithelium, hemorrhage, necrosis, glandular destruction, and frequently infection. In the healing of ulcers, irrespective of the way they may have been produced, the basic features consist in development of protective connective tissue by hyperplasia, with resultant scar.

The experiments of Bolton and of Rosenow are particularly interesting. The latter investigator has recently revived the bacterial theory of ulcer causation. By intravenous injections of streptococci usually obtained from throat and mouth lesions which have undergone a certain mutation culturally, Rosenow has succeeded in producing both acute and chronic gastric ulcer. From these ulcers he has been able to recover organisms, morphologically and culturally, similar to those with which, experimentally, he produced the lesions. He also has been able to demonstrate the inter-relationship of infections of the appendix, gall-bladder and

pancreas, and peptic ulcer with regard the streptococcus. He has shown the possibilities of organisms infecting the mouth, throat, or head sinuses gaining access to the general circulation and contributing to intra-abdominal pathology, or, by direct infection of the alimentary tract producing or aggravating ulceration. While Boetcher, Letulle, Dieulafoy and others many years ago advanced similar experimental proof, yet Rosenow, by tissue culture, has emphasized the significance of bacterial action in association with local or disseminated foci of chronic infection. However, as the definite causative factor of gastric ulcer, and particularly with respect its being *only cause*, Rosenow's bacterial proof cannot be wholly accepted. The ulcers which he has produced are not in any way dissimilar from those produced by other investigators using organisms other than the streptococcus or those ulcers caused by a wide variety of non-bacteriological agents. Rosenow has failed to show that the streptococci recoverable from injured mucosæ in the alimentary tracts of his experimental animals have not been secondarily deposited there as a consequence of some preceding, local, cellular fault. Control experiments using organisms other than streptococci are likewise lacking.

Antedating Rosenow's observations, and apparently being more basic in principle etiologically, are those of Bolton. This investigator has shown by an extensive series of experiments that *non-bacterial* tissue injections quite uniformly produce all grades of peptic ulcer in susceptible animals. Sterile emulsions of the gastric mucosa, of the appendix, gall-bladder, or liver injected intravenously or intraperitoneally produce in animals a toxic serum. At the end of from ten to fourteen days this serum has such selective affinity that its introduction into the circulation of animals produces necrosis and ulceration in the organs from which the original cell emulsions were made. The toxicity of the serum persists and may produce its effects after more than a year. The serum gives the various test-tube reactions for immune bodies, namely, lysins and precipitins. The most pronounced reactions are with gastric cell emulsions. Their injection rapidly causes acute hemorrhagic ulcers, which ulcers may later become chronic. Saturation of the serum with gastric cells before injection, robs the serum of its toxic qualities. Bolton has called this immune serum "gastrotoxin."

Bolton's gastrotoxic serum appears to indicate that, as result of a wide variety of ailments or systemic constitutional affections, disintegration of body cells may occur. The initial agents causing the malfunction form a great and dissimilar group. The poisons elaborated are of specific nature with respect the tissue from which they are derived. It would seem that the demands constantly being made upon the stomach result in a great cellular waste. The cellular waste satisfies the essentials of a protein poison. When

opportunity occurs this poison is capable of producing local cell changes in the gastric mucosa, which changes furnish the *anlage* for a subsequently developing ulcer. Once this initial local injury has been done to the gastric lining, the autodigestion of the mucosa by gastric juice or its ability to harbor bacteria are possible, and *not until then.*

The above brief summary indicates that we cannot expect to ever establish a single definite cause of gastric ulcer. The immediate causes of ulcers vary widely and are highly individual. The essential point is that through some malfunction a point of least resistance occurs in the gastric lining and *normal* gastric physiology is permitted to produce an *abnormal* tissue change. *It would likewise appear that we have no basis for regarding gastric ulcer as a distinct disease entity.* It seems more proper to consider that ulceration of the gastric mucosa is a local accident in association with a systemic upset, the nature of which upset may be extremely varied. When once the damage has been done locally in the stomach wall, whatever may be the primary cause, the future course of the process is similar. Pepsin and hydrochloric acid attack the inert gastric cells, necrosis results and ulcer is established. The course of the process depends mainly upon the persistence of the underlying systemic causes, variation in the quality of the gastric juice, local intragastric trauma (as by food, bacteria etc.), and the influence of the intrinsic muscles forming the stomach wall. Such facts strongly suggest that *peptic ulcer presents aspects indicating its being a self-limited disease.*

3. *Histopathological.* From the inception of symptoms clinically, it is impossible to prognosticate the future course of any peptic ulcer. While many ulcers destroy relatively little gastric mucosa and produce harmless scarification, thus evidencing a tendency to spontaneous cure, other ulcers, seemingly equally inocuous clinically, progress rapidly through various pathological stages and are very shortly associated with stenoses, extensive callous formation, hemorrhage, perforation, or malignancy. However, chronicity in the histopathological sense by no means indicates that a peptic ulcer is old in terms of months or years. Malignant change in ulcer edges may occur quickly and in no way point to the long existence of a previously benign affection. It is of much significance to observe variation in the appearance of pathological transformations in ulcers situated similarly in various parts of the stomach. While fully 85 per cent. of all gastric ulcers occur in the pylorus, antrum, and along the distal four-fifths of the lesser curvature, and while in these positions the greatest damage can be done by ulcer, yet just as striking changes occur in ulcers situated in other portions of the viscus. It is of importance to note that fully two-thirds of the gastric ulcers occur in the portion of the stomach whose mucous membrane is not devoted to the elaboration and secretion of hydro-

chloric acid. It would also seem to be of significance to recall that the majority of peptic ulcers occur in that part of the stomach in which the greatest circulatory, muscular, and nervous activity is manifest; that the fewest ulcers occur in that portion of the viscus which is fixed and which has to perform mainly the duties of a food receptacle and an acid-secreting organ.

4. *Physiological.* (*a*) *Chemical.* It is a striking commentary upon the empirical nature of standard treatments of peptic ulcer that they have as their basis the principle that gastric chemistry has been greviously upset. It has been supposed that this disarrangement has been caused either by the development of ulcer or that the upset itself has produced the ulcer. This chemical disturbance is generally put down as being in the nature of an overproduction of acid gastric juice and pepsin. It is presumed that the so-called "corrosive effects" of this overactive digestive juice produces gastric ulcer or prevents healing of one already present. It is significant to observe, in the first place, that the normal gastric juice frequently varies in strength well beyond the range of the so-called increases which are often demonstrable when gastric ulcer exists. In a recent study of 500 cases of peptic ulcer, in but 40 per cent. did I find a free HCl concentration above 0.3 per cent. In 35 per cent. the acidity was well within the "normal" range, and that in the remaining 25 per cent. the acidity was reduced or was entirely absent. Similar observations have been recorded experimentally by Carlson, and in man by Rehfuss and his coworkers. Moreover, the portions of the stomach which are least the site of gastric ulcer are those in which, physiologically, the concentration of hydrochloric acid is greatest and where the acid is in nascent form. That there are causes of gastric ulcer other than the variation in the hydrochloric acid concentration is also supported by the observation that in bile-free stomachs large amounts of hydrochloric acid in the concentration of as high as 0.6 per cent. mixed with pepsin may be placed in the stomach and yet not produce ulcer. It is also evident that the primary damage which results in gastric ulcer is not dependent wholly upon acid gastric juice by the ancient observation that if this were the case the stomach would digest itself. Clinical and histopathological evidence indicate that when once the initial damage to the gastric lining has been brought about from whatever cause, digestion of partly devitalized or necrotic mucous membrane may occur with equal readiness in the presence of normal or even subnormal gastric acidity. With these facts in mind it seems strange that treatment of gastric ulcer has largely rested upon a chemical foundation, which chemical foundation was extremely unstable and shifting. It has been presumed that in ulcer stomachs, overacidity was present, and that this overacidity must be counteracted by alkalinization before healing could take place. It has been presumed that the pain of gastric

ulcer existed as a consequence of irritation and "corrosion" of raw mucous surfaces by gastric juice rich in hydrochloric acid and pepsin even though the researches of Pawlow, Rehfuss, Carlson, Hertz, Hamburger and others have demonstrated that pain is not uniformly present when gastric acidity is highest, and that in known ulcer cases the introduction of high percentages of acid fails to produce pain. The relief from pain in gastric ulcer cases is quite as prompt when alkalies are administered in low acid cases as when such are used in high acid cases. Prompt relief of pain in such instances is also secured by the exhibition of lavage, alcohol, opiates, diet, etc. It would seem, therefore, that medical treatment of peptic ulcer based upon chemical upsets supposed to be associated with the ailment presents much that is empirical, irrational, and unscientific.

(*b*) *Physiological Motor Consideration.* Modern physiological investigations have been especially rich in elucidating the motor activity of the stomach under normal and pathological conditions. For this knowledge we are indebted to the development of the roentgen-ray and the tireless researches of Pawlow, Cannon and his pupils, and to Carlson. These investigators have established the significance of certain gastric motor cycles. They have shown that these cycles occur with almost mathematical precision. Upon these constants it would appear to be possible to base clinical observations and therapy. It would also seem that the mechanical factors concerned with digestion are of greater importance than are variations in the secretory function. It is with the mechanical features in digestion that we purpose to deal in suggesting a rational régime in the treatment of peptic ulcer. In order to emphasize the points mentioned below in detailing the type of treatment which we advance, the chief facts established by physiological investigation with regard the mechanical activity of the stomach now will be summarized:

The fasting stomach is in a state of tonic contraction. It is rarely empty. Its contents contain both hydrochloric acid and pepsin. These secretions apparently aid in preserving gastric tone, in digesting mucus, dead bacteria, and desquamated cell detritus. Hunger is manifested by rhythmic gastric systoles. These precede appetite desire and cause an unpleasantness that leads to eating. Repeated swallowing motions cause inhibition of gastric tonus. Eating starts gastric-juice secretion. Food entering the stomach initiates the peristaltic movements peculiar to gastric digestion. These peristaltic movements continue so long as food remains in the stomach. They pass from the pars media toward the pylorus in rhythmic sequence in a given case with equal intensity, thus maintaining a constant pressure in the antrum. The proximal third of the stomach acts mainly as a sac or reservoir and is comparatively free from peristaltic activity. The acid reaction of con-

tents in the fundus of the stomach closes the cardia. The discharge of chyme from the stomach is intermittent. The pylorus opens only when acid gastric contents relax the sphincter. The presence of acid chyme in the duodenum closes the pylorus and keeps it closed until the duodenal juices have rendered its contents neutral or alkaline. Acid chyme stimulates the flow of pancreatic juice and bile. The peristaltic waves press acid chyme toward the pylorus and intimately mix gastric contents and digest them. Only after duodenal contents have become neutralized can the pylorus relax and acid chyme pass through. This alternate opening and shutting of the pylorus with discharge of chyme is continued until the stomach is empty.

Gastric Secretion and Gastric Emptying are Greatly Influenced by the Kind of Food Ingested. Water and normal salt solution cause limited gastric secretion and rapid stomach emptying. This rapid exit causes but feeble peristalsis. Carbohydrate foods leave the stomach quickly because on account of their failure to unite with acid gastric juice they permit a large amount of free acid to come into contact with the pyloric sphincter and thus conduce too rapid opening of the pylorus. Marked retardation in the discharge of carbohydrates occurs if such be mixed with alkaline solutions. The alkali delays the appearance of acid by checking temporarily the secretion of acid gastric juice and also by uniting with the free acid already poured out. The acid control of the pylorus is thus interfered with and emptying is delayed. Protein food leaves the stomach slowly because proteins join with free HCl and thus for a time retard the development of an acid reaction which initiates pyloric opening. Protein has also been shown by Khingine to cause the secretion of 50 per cent. more gastric juice during the first four hours of digestion than when carbohydrate is fed. When such protein is passed into the duodenum there is more acid to neutralize than when carbohydrate was fed, consequently the pylorus remains closed for a longer time and the antrum of the stomach is subjected to intense peristaltic activity without pyloric relaxation. It is estimated that in such event the peristaltic waves number from 300 to 500 per hour. Cannon states that at the end of a half-hour eight times as much carbohydrate as protein has been absorbed, and there exists twice as much carbohydrate as protein in the jejunum. While carbohydrates begin to leave the stomach at once, proteins are delayed from one to three hours. Fats remain longest in the stomach because they excite little free HCl production and consequently the acid control of the pylorus is reduced to a minimum.

Vigorous mixing of food with acid chyme occurs in the antrum and pylorus, the zone in which 60 per cent. of all gastric ulcers are found. Food delay in this locality permits of increased local movement of the viscus, greater opportunity for food and acid to remain in intimate contact with injured gastric lining, hence, opportunity

for the maximum of trauma, infection, digestion of damaged tissue, stress on the pyloric sphincter, and local alterations in circulatory and neuromuscular mechanism.

The facts which I have enumerated have been established as constants by repeated laboratory and clinical investigation. In my opinion they furnish a logical ground-work for the treatment of peptic ulcer. They place such treatment upon a rational basis and remove from it many purely empirical and "rule-of-thumb" features which characterize commonly accepted modes of therapy. I am convinced of its clinical usefulness after five years of careful clinical observation.

5. *Selection of Cases for Types of Treatment.* The principles involved in treatment of gastric ulcer, of whatever nature, first demand *search for and eradication of the primary systemic fault.* Often the localization of such is difficult. Inasmuch as foci of infection may exist in oral adenoid tissue, head sinuses, about teeth or in systemic lymph gland chains, these must be removed promptly. Intra-abdominal infections must likewise be eradicated, *e. g.*, diseased appendix, gall-bladder, Fallopian tubes, ovaries, ulcers, or subinfections of the bowel. It would appear quite inadequate to remove external local foci of infection and to leave behind intra-abdominal foci containing bacteria already accustomed to their environment and ready to spread their operations to the gastric lining when opportunity offers. It would appear that the removal of these variously situated germ centers constitutes a fundamental step toward the cure of gastric ulcer.

After local foci of infection have been removed the mode of treatment is further influenced by the *type* of ulcer that has been proved to exist. Unless ulcers with much scar or causing great gastric deformity are demonstrated to be luetic, little hope of permanent relief by medical measures can be offered. Surgery promises the greatest prospect of relief to such cases. Intense pain, frequent hemorrhage, perforation, or the danger of malignant change taking place in calloused ulcers likewise contra-indicate non-operative care. Unfortunately we have no clinical or laboratory tests which indicate to us *what type* of ulcers will become malignant or *when* early malignant change is taking place. The roentgen demonstration of calloused ulcer exceeding 2 cm. diameter when such is associated with history of frequently recurring ulcer symptoms and positive chemical test for blood constantly determined in the stools, forms a clinical hint that malignancy can be expected. The most competent clinicians are agreed that calloused, recurring ulcers, located in the pyloric end of the stomach should be treated operatively. Excision should be performed when mechanically possible. If excision is impracticable, then infolding, or cautery puncture, with or without gastrojejunostomy, yields the most satisfactory results. In non-obstructing ulcers, gastrojejunos-

tomy should always be accompanied by permanent pyloric closure. Gastrojejunostomy properly performed acts by aiding gastric emptying, by diminution of free HCl (an average of 18.2 points in 196 consecutive cases in our series), and by permitting limited jejunal regurgitation into the stomach.

Successful medical treatment of ulcer thus first demands careful selection of cases to be so treated. It is indicated only in ulcers associated with little callus, or if calloused, located in portions of the stomach in which stenoses are not liable to result or where surgical procedures cannot be carried out. Certain essential principles are to be borne in mind in carrying out medical treatment.

II. Outline of Author's Method of Non-surgical Treatment.

1. *Rest in Bed.* Both physical and mental for from one to three weeks. Bodily and psychic activity stimulate peristalsis.

2. *Rest to the Stomach Itself.* When it is recalled that during an ordinary meal the digestive processes demand more than 2000 peristaltic waves, the effect of such as a mechanical irritant to an ulcer or the ulcer-bearing area cannot be disregarded. Complete rest for the stomach also demands avoidance of irritating medicine, gastric lavage, and frequent abdominal examinations of the suspected focus.

3. *Local Applications to the Abdomen.* Painful spasms are further prevented by having constantly applied to the abdomen compresses saturated with Ochsner's fluid (alcohol and boracic acid.)

4. *Keeping the Stomach Empty of Food.* This promotes healing by limiting local irritation from the food itself, from reducing the amount of gastric juice required to digest food, by limiting gastric peristalsis and avoiding painful gastrospasms which limit free circulatory interchange. The abstinence from food by mouth should be insisted upon for from three to seven days, according to the case. The period of fast is determined best by clinical disappearance of gastric spasm (pain, regurgitation, waterbrash, heartburn) and by fluoroscopic proof of absent or diminished gastric peristalsis. During the fast *paraffin wax is chewed* for fifteen minutes every hour. It keeps the mouth clean, promotes free flow of protective saliva and mucus, counteracts painful hunger contractions and gastrospasms, and allays thirst.

5. *Rectal Feeding.* During the fasting period, rectal feedings are instituted. From 500 to 1000 calories of nutrient mixture are given in twenty-four hours. We use a clystra containing 1 ounce of 50 per cent. alcohol, 1 ounce of glucose with normal salt solution to make 240 c.c. The nutrient enema is given at body temperature by the drop method. The drops flow at the rate of 30 to 60 drops per

minute. During the first day of rectal feeding, gtt. x of tr. opii are given with each enema.

6. *When Mouth Feeding is Begun.* Usually from the fourth to seventh day. Two factors control the choice of diet: (*a*) nourishment should be liquid and administered warm in small quantities frequently, and (*b*) carbohydrates should be selected.

(*a*) Small quantities of liquid food should be frequently administered in order that the stomach empty rapidly with the least effort and thus remain food-free for the longest time, thereby giving maximum time of rest for ulcer healing. The duodenal digestion must be called upon until gastric conditions warrant demands being made upon stomach digestion. Keeping the stomach food-free keeps hydrochloric acid or pepsin production to a minimum. From 4 to 6 ounces of warm liquid are given every hour.

(*b*) As experimental facts have established, carbohydrate foods leave the stomach most quickly. Therefore, liquid carbohydrate mixtures (barley water, rice gruel, thin cream of wheat, thin creamed vegetable soup, etc.) are fed. Milk is not given as routine. Milk results in almost pure protein clots in the stomach. These act as do other proteins and remain for a long time in the stomach as a source of irritation, as stimuli to acid secretion and as choice culture media for bacteria. If milk be given at all, it should be first parboiled or predigested. Carbohydrate liquids produce the least secretion of HCl and pepsin and are weak stimuli of gastric peristalsis and impose the minimum of work upon the duodenum. It should be recalled that the pylorus opens only when the duodenal contents are neutral or alkaline. If the gastric contents are of such nature as to impose slight demands upon the stomach secretions and motility, the duodenum has little work as a neutralizer to perform and the pylorus remains free from spasm and opens readily. There are thus avoided gastric stagnation and accumulation of distressing free and combined acids, which prevent healing and which usually demand frequent lavage or the exhibition of large quantities of alkali.

7. *Limitation of Overproduction of Gastric Acid.* This is obtained by keeping the stomach food-free as above described. This secondarily limits both the frequency and the strength of gastric peristaltic waves. Unless food leaves the stomach rapidly, gastric glands continue secretion and coincidently stress of gastric peristalsis upon the pylorus keeps up constant irritation of ulcer-bearing areas.

If the above points established by modern physiological research are borne in mind, the exhibition of large quantities of alkali are unnecessary. Their use is certainly unscientific. Providing the gastric lumen is patent, the stomach empties freely. There is no stagnant, irritating, fermenting residue. Large quantities of alkali, according to Pawlow and to our clinical and laboratory experience, create pernicious increases of gastric acid and of mucus and generally

demand relief by lavage. Moreover, we have shown that many gastric ulcer cases do not exhibit hyperacidity or hypersecretion. It is true that the stomach *can* neutralize large quantities of alkali if compelled to do so, but there is no physiological reason why it should be called upon to thus overwork. It will be remembered that the normal habitat of gastric epithelium is an acid or at the best a neutral medium. If these epithelial cells are called upon to live in an excess of alkali, they live, as it were, in the presence of a foreign body. Experiments in artificial tissue growth have shown that cell proliferation is retarded by hyperisotonic alkaline solutions. Hence, overalkalinization may prevent healing. Attempts at protection from this foreign body (excess alkali) are shown (1) by the acid-producing glands oversecreting, and (2) by the mucoid degeneration of physiological fatigue which results in the throwing out over the secretory glands of a protective layer of mucus. The vicious circle thus formed results in enormous secretion of acid and mucus and is doubtless at least a partial explanation of the so-called hypersecretion associated with gastric ulcers, particularly when such are treated by the overalkalinization method. To combat this condition of affairs the patient's stomach must be washed frequently or greater quantities of alkali must be given in order to overpower the stomach's defensive mechanism and produce fatigue or exhaustion of the acid-secreting mechanism. It is a common observation that those patients who are treated for ulcer by the overalkalinization procedure always require frequent gastric lavage in order to ensure their comfort. This frequent lavage is to be condemned not only on account of its disagreeable features but because it acts contrary to the primary requirement of healing, namely, *rest of the affected part*. It is quite evident to those who have watched the behavior of a stomach by means of fluoroscopic screen when a tube is inserted into it that gastric lavage defeats this primary principle of healing. Lavage is generally accompanied by vigorous gastric contractions that persist not only during the maneuver but often for a long time afterward. If dieting is arranged on the carbohydrate basis, alkali is given in only sufficient quantities to keep the stomach slightly acid or neutral and to neutralize the duodenum, thus aiding in pyloric relaxation, gastric lavage need rarely be instituted during the entire course of a patient's treatment. In the past five years we have not employed lavage therapeutically in ulcer cases more than a dozen times. Lavage is so rare a procedure in my clinic that my associates and patients consider such an order as almost contra-indicated. It is readily judged how a treatment of which lavage does not form a prominent feature contributes much to a patient's peace of mind and shortens the period of hospital incarceration.

8. *Medical Treatment.* It is doubtful if any form of medicine has a direct healing effect upon peptic ulcer. Medicines are adminis-

tered largely to counteract discomfort due to three main causes, namely, (*a*) painful gastrospasms, (*b*) accumulations of overacid gastric contents associated with peristaltic unrest, (*c*) pain associated with perforation.

(*a*) *Painful gastrospasms* are usually controlled by carrying out the dietetic principles which I have above mentioned. The chewing of paraffin wax relaxes the pyloric spasm largely through stimulating a proper swallowing reflex and by fatigue of hunger-like contractions. Certain types of case in which there is an individual vagus hypertonia, or when ulcers are located at or near the orifices, demand the exhibition of antispasmodic medicines, such as atropin, tincture of belladonna, or bromides. In the early stages of the treatment, when the stomach is being kept as free as possible of contents, atropin may be given hypodermically or bromides may be placed in the nutrient enemata. Later, when food is being given by mouth, tincture of belladonna in doses of from 5 to 15 drops may be administered fifteen minutes before feeding from three to six times daily. We have not found useful, as analgesics, the exhibition of large doses of such "protective" medicines as bismuth and olive oil. These medicines doubtless act by affecting the rate and intensity of peristalsis, although they may have some effect in proved cases by direct action upon the ulcer. At times orthoform, given in 10-grain doses, in warm water, is an efficient local anesthetic when it is able to come in direct contact with an open ulcer.

(*b*) *For the relief of overacid gastric accumulations*, sodium bicarbonate is contra-indicated, because its administration results in the production of annoying accumulations of carbon dioxide with resultant gastric retention or painful belching, and because its neutralizing value is comparatively low. Large quantities of bicarbonate of soda are necessary to give relief, and the administration of such secondarily produces excessive gastric secretion. If alkalies are indicated, better results are obtained by the exhibition of frequent small doses of milk of magnesia or calcined magnesia. The ordinary case is very comfortable when from five to ten grains of calcined magnesia are given every two or three hours. Many cases require no exhibition of alkali if the physiological principles above outlined form the basis of the treatment. Only in very extreme cases is it necessary to employ gastric lavage. When it is employed, warm Carlsbad water (1 dram of artificial Carlsbad salts to 1 quart of water) may be satisfactorily administered. Usually the exhibition of atropin or belladonna for the relief of gastrospasms exerts a definite effect toward controlling oversecretion of acid gastric juice.

(*b*) The acute prostrating *pains of perforation* are best controlled by the prompt administration of morphin hypodermically, rest in bed, and hot compresses to the abdomen. Only prompt surgery saves the patient's life.

9. *Hemorrhage.* Constant seepage, demonstrated either microscopically or clinically, is generally an indication for abdominal section. Intermittent seepage may be controlled best by rest in bed, morphin hypodermically, intravenous injections of fresh horse serum, coagulose, or by copious transfusion of whole blood. In acute hemorrhage accompanied by vomiting, prompt lavage of the stomach with water at 110° F. frequently stops both the vomiting and the hemorrhage. The exhibition of morphin, rest in bed, and whole blood transfusions generally prevent recurrence of hemorrhage. In this class of case, however, surgical intervention should be resorted to early, and this especially if in a given patient, frequent, copious, prostrating hemorrhages occur.

10. *Bowels.* During the early periods of treatment, simple soapsuds enemata may be administered every second day. After the second week, morning doses of phosphate of soda or Carlsbad salts in hot water may be applied. In chronic cases, liquid paraffin given in equal quantities of warm cream result in easy motions, and the paraffin appears to have certain protective value upon the ulcer bearing area.

Summary of the Author's Dietetic Régime in Treatment of Peptic Ulcer.

Days 1 *to* 7 (Time varies as outlined in above description of treatment).

By Mouth. One-half ounce warm water hourly when awake. Patient chews paraffin wax for fifteen minutes at least once in two hours. Juice of sweet orange or grape fruit occasionally.

By Rectum. Nutrient enema consisting of 50 per cent. alcohol 1 ounce, glucose syrup 1 ounce, and normal salt solution 6 ounces every four hours. The enemata are preceded by a cleansing irrigation of the colon with normal salt solution. They are given at body temperature by the drop method at the rate of from 30 to 60 drops per minute. Calories daily approximately 1000. During the first two days, tr. opii ♏x is added to each second enema.

Days 3 *to* 14 (case of average severity).

By Mouth. From 4 to 6 ounces of water gruel at temperature of 100° F. The gruel is taken slowly through a glass tube. Gruels are made from rice, cream of wheat, oatmeal, sago, corn-meal, malted milk, macaroni and vermicelli, rusks, potato, asparagus, cauliflower, beans, peas, and boiled onion. They are strained before feeding. Flavoring with coffee, chocolate, vanilla, caramel, etc., renders the cereal gruels palatable and their administration easier. To the vegetable gruels small quantities of arrowroot or corn-starch are added to secure a thin emulsion.

Before each feeding, paraffin wax is chewed for five minutes. Warm water or sweet orange or grapefruit juice are allowed as desired, but never in greater quantity than 1 ounce at a time.

By Rectum. During the first two days of mouth feeding, two alcohol-glucose-saline nutrient enemata are given. During the second two days, *one* such nutrient enema is administered. After the fourth day of mouth feeding no rectal feedings are given in the average case. Calories approximately 800.

Days 14 to 21.

6.30 A.M. A glass of hot water and 1 teaspoonful of non-effervescent sodium phosphate.

7.30 A.M. One ounce of sweet orange or grapefruit juice, 2 ounces of thin cream of wheat, or farina, or well-cooked rice, or corn-meal, 2 ounces of skimmed, parboiled milk, may be taken with cereal, and if desired a small quantity of powdered sugar used; 1 zweibach with a thin layer of fresh butter, 4 ounces of parboiled skimmed milk, containing half volume of lime-water, served warm and flavored with coffee, cocoa, caramel, or vanilla.

9.30 A.M. Six ounces of thin water gruel from cereals or fresh vegetables, strained and served hot, 1 rusk or zweibach, or dry toast.

11.30 A.M. Four ounces of malted milk, whipped egg, with parboiled milk, corn-starch pudding, simple custard lightly cooked.

12.30 P.M. Six ounces potato, pea, bean, or asparagus puree, (strained), or vegetable broth; 4 ounces of salisbury steak (moderately well cooked) to chew; 2 ounces (cooked weight) of thin rice, sago, tapioca, or corn-starch pudding made with parboiled milk and egg yolk; 2 ounces of parboiled milk and small quantity of pulverized sugar may be eaten with the pudding; 1 rusk or zweibach, 6 ounces of parboiled milk and quarter volume of lime-water flavored to taste.

4 P.M. Four ounces of water gruel from cereals, 1 very soft poached egg, 1 rusk or zweibach, 4 ounces of hot Vichy water.

6 P.M. Four ounces of whipped egg, 2 rusks or zweibach, 6 ounces of malted milk (thin), flavored to taste, or cereal water gruel or parboiled milk and quarter volume of lime-water gruel.

9 P.M. Six ounces of water cereal gruel or 4 ounces of malt marrow, 2 graham crackers. Calories approximately 1500.

Days 21 to 42.

6.30 A.M. Two teaspoonfuls of phosphate of soda in a glass of hot water.

8 A.M. Juice of 1 sweet orange or half sweet grapefruit, or boiled prunes passed through fine collander; 2 ounces (cooked weight) of thin cereals (cream of wheat, farina, oatmeal, corn-meal) 2 ounces of skimmed milk and small amount powdered sugar, 1 soft poached egg, 2 zweibach, 2 rusks or 2 thin slices of well-toasted graham bread, 1 pint of hot skimmed milk + a quarter volume of lime-water flavored to taste (cocoa, vanilla, etc.).

10 A.M. One pint of hot parboiled whole milk and fifth volume of lime-water, 2 rusks or graham crackers.

12.30 noon. Four ounces of creamed soup from vegetables, strained, 6 ounces rare meat to chew, 4 ounces well-mashed potato or baked potato (mealy inside), or carrot, peas, beans, cauliflower, Brussels sprouts, or asparagus (all vegetables passed through a strainer and served with 15 grams of butter), 4 ounces (cooked weight) of pudding from rice, corn-starch, sago, tapioca, cream of wheat, or farina or 4 ounces of custard, pulp of sweet orange, grapefruit, or prune whip, or chew 6 ounces of watermelon or cantaloup, half pint of hot skimmed milk.

3.30 P.M. One hundred and fifty c.c. of hot whole milk and quarter volume of lime-water or 150 c.c. of malted milk or weak cocoa.

6.30 P.M. Two rusks or zweibach or 2 slices of well-toasted graham bread, 2 very soft poached eggs, 100 grams of sweet apple sauce or 1 baked apple (omit skins), or juice of sweet orange or half of grapefruit, or chew 6 ounces of melon, 1 pint of skimmed milk, hot.

9 P.M. Two hundred and fifty c.c. of whole parboiled milk and quarter volume of lime-water of 250 c.c. of malted milk, hot. Calories for twenty-four hours approximately 2000.

General Diet after Three Months. If distress, patient should go back to 7 to 21 day diet.

7 A.M. One pint of skimmed milk and half-gill of cream.

9 A.M. Two pieces of toast without butter, juice of one sweet orange or grapefruit or ripe melon or apple sauce or baked apple (do not eat skin) or marmalade, 1 dish or well-cooked cereal (oatmeal, farina, or cream of wheat), 2 very soft poached eggs, 2 cups of hot, sweetened water. The water may be made more palatable by flavoring with cocoa, tea, coffee, or cream.

11 A.M. One cup of bouillon (two cubes), two graham crackers.

1 P.M. This should be the heavy meal of the day. It may consist of meat (rare beef, rare hamburger steak, lamb, or white meat of fowl), fish (never fried), oysters, well-cooked spinach, cauliflower, carrots, squash, peas, (hulled), string beans, Brussels sprouts, baked or mashed potatoes (in moderation), rice with gravy, simple puddings made from cereals, corn-starch, gelatine, well-cooked fruit sauces, simple cakes, no white bread (all bread should be made from dark flour and should be at least one day old), 1 pint of skimmed milk taken hot.

4 P.M. One glass of hot peppermint water (20 drops of "essence" of peppermint to the glass), sweeten to taste and drink slowly; two graham crackers.

7 P.M. A light lunch consisting of vegetable soup, simple salad, toast, soft eggs, and plain puddings or cake, with or without ripe cooked fruit sauces; 1 pint of hot skimmed milk.

Bedtime. One glass of malt marrow, malted milk, or hot skimmed milk. Calories approximately 3500 for twenty-four hours.

THE OPERATIVE TREATMENT OF HYDROCEPHALUS: A PRELIMINARY REPORT OF FORTY-ONE PATIENTS.[1]

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The treatment of hydrocephalus has been most discouraging. One operative method after another has been advocated as a possible means of successful drainage in these patients; one or more cases are reported and then nothing more is heard of the method, the patients having died and the method no longer used. Frequently a "new" method of drainage is published more upon a theoretical basis rather than upon practical applicability; one case alone is published as having been operated upon and the result was death; surely, we must grant that the method may have been new, but the result has been the same as in the earlier methods of treatment, and unless the percentage of cures, or even improvement, can be raised, such methods are naturally soon discarded.

The chief cause of failure in these methods of drainage is, in my opinion, not due so much to the ignorance regarding the nature of hydrocephalus itself, what the cerebrospinal fluid really is, its method of secretion and excretion and similar points of almost total obscurity, but rather to a lack of appreciation of the fact that the condition of hydrocephalus is rarely limited to a dilatation of the ventricles alone—that is, the so-called hydrocephalus interna, which is the result of a blockage of the escape of cerebrospinal fluid from the ventricles into the subarachnoid spaces by an obstruction of the adequate of Sylvius or of the foramina of Majendie and Luschka; but, on the contrary, the condition is most frequently due to a lessened excretion of the cerebrospinal fluid through the subarachnoid cranial and spinal veins, the sinuses, possibly lymphatics, etc., so that the type of so-called hydrocephalus externa is developed. It is this latter condition of so-called hydrocephalus externa that is most frequent and yet the methods of treatment have been almost all of them directed to a drainage of the ventricles in the condition of hydrocephalus when it is a fact that the ventricles do not require drainage in only a small percentage of these cases; they are not dilated in the majority of cases because the aqueduct of Sylvius and the foramina of Majendie and Luschka are not obstructed, and there is a free escape of cerebrospinal fluid from the ventricles into the subarachnoid spaces, so that any operation merely directed to connect the ventricles with the subarachnoid or subdural spaces in those cases is of no possible benefit. In the comparatively rare cases, however, of true hydrocephalus interna due to a definite

[1] A complete report of the cases in detail will be published later.

blockage of the aqueduct of Sylvius or of the foramina of Majendie and Luschka by a subtentorial lesion, those methods of drainage are logical, and yet I believe they are of very limited usefulness in that the condition of hydrocephalus interna is almost always, in my opinion, the result of an early diffuse meningitis, the results of which (adhesions, exudate, thickenings, etc.) may not only block the escape of cerebrospinal fluid from the ventricles into the subarachnoid spaces, but also so impair and even completely block the channels of escape of the cerebrospinal fluid through the cortical veins, sinuses, possibly lymphatics, etc., so that even if the ventricles were connected with the subarachnoid spaces, yet the cerebrospinal fluid would not be drained from the cerebrospinal canal; that is, the condition of blockage of the escape of cerebrospinal fluid is a diffuse one rather than a mere obstruction of the ventricles; there are cases, to be sure, of hydrocephalus interna due to a tumor or cyst formation blocking the aqueduct of Sylvius or the foramina of Majendie and Luschka, and where the ventricular subarachnoid method of drainage would be logically applicable, but these cases form but a very small percentage of the large group of hydrocephalus patients; in this series of forty-one patients, twenty-six were of the external type, whereas only fifteen were of the internal type of hydrocephalus; besides, the method of draining the cerebrospinal fluid described in this paper can be used in either condition—that is, whether the condition is one of the internal or of the external type of hydrocephalus. The condition of unilateral hydrocephalus may be mentioned as a very rare occurrence; it is due to a blockage of the aqueduct of Sylvius and the opposite foramen of Monro; one of our patients was of this type.

In order to differentiate whether the condition is one of hydrocephalus interna or of externa, or of both (and this last condition occurs fairly frequently), the method of Dandy and Blackfan[2] in their excellent monograph can be used whereby the time of excretion of phenolsulphonaphthalein first from the ventricles and then from the spinal subarachnoid spaces can be observed and the amount estimated in the urine; if practically of equal duration and amount then the ventricles are not blocked and the condition is one of hydrocephalus externa, or of both, whereas, if the excretion of phenolsulphonephthalein injected into the ventricles is greatly delayed and lessened in amount in the urine, then the ventricles are blocked and the condition is one of hydrocephalus interna. A much more simple method, however, can be used, and I believe it is of sufficient accuracy to warrant its use whenever a differentiation of the two conditions is desired in the treatment of hydrocephalus. I make this statement because the operative method to be described in this paper makes such a preliminary test unneces-

[2] Am. Jour. Dis. Child., December, 1914, pp. 406–482.

sary, as the differentiation of the internal from the external type of hydrocephalus can be ascertained at the time of the operative procedure and treated accordingly through the same operative incision and at the same time. If, however, it is desired to ascertain the type of hydrocephalus before the operation, then a ventricular puncture needle can be inserted into the ventricle through a very small skin incision at the anterior fontanelle as far from the median line as possible, and at the same time a spinal puncture needle is inserted into the lumbar subarachnoid space; with the patient lying upon his side, the median line of the head being upon an exact level with the spinal canal and the patient being perfectly quiet, the pressure and rate of flow of the cerebrospinal fluid from both the needles should be the same if the ventricles and subarachnoid spaces are in free communication; the condition would therefore be one of hydrocephalus externa. If, however, the ventricles are blocked, then the pressure and rate of flow of cerebrospinal fluid from the ventricle needle is greatly in excess of that from the spinal puncture needle, which may be but a few drops; the condition therefore would be one of hydrocephalus interna. Another aid in the differentiation in the older children may be found in the roentgen-ray—the internal type producing convolutional markings throughout the vault of the skull, whereas this would naturally not occur in hydrocephalus externa.

Many operative procedures have been used in the past to combat the condition of hydrocephalus. In the early days of Hippocrates merely repeated lumbar punctures and ventricular punctures were performed, but the result was invariably the same—a steady advance of the condition and frequently a much more rapid progress following the tapping, as though the sudden removal of the increased amount of the cerebrospinal fluid stimulated in some way a greater secretion of the cerebrospinal fluid so that the condition rapidly became worse. Occasionally, however, a case has been reported improved by such treatment; such cases, however, were rare and the reports of them were seldom made after one year had elapsed; they may have been, therefore, merely temporarily benefited. Drainage of the cerebrospinal fluid externally to the skin has almost always resulted fatally on account of the consequent infection and the resulting meningitis.

For these reasons many complicated and most tremendous operations have been devised to drain successfully the cerebrospinal fluid from the ventricles and the subarachnoid spaces, and all of them have been most disappointing.

Draining the cisterna magna (fourth ventricle) into the adjacent sinuses or into the pleural cavity by means of strands of various material through a suboccipital opening; connecting the spinal subarachnoid canal with the retroperitoneal space by means of a laminectomy and a laparotomy (and naturally limited to cases of

external hydrocephalus); joining the third ventricle to the subarachnoid spaces by means of a tube through the corpus callosum and the various other methods described in the literature—usually these methods have been abandoned after a few patients have been operated upon with the usual result—little or no improvement and only too frequently the death of the patient. Naturally, the condition is a most serious one and even the most dangerous operative procedures can be considered; no operation, however, should be advised to drain the ventricles unless it is definitely known that the ventricles are blocked and that the condition is not one of hydrocephalus

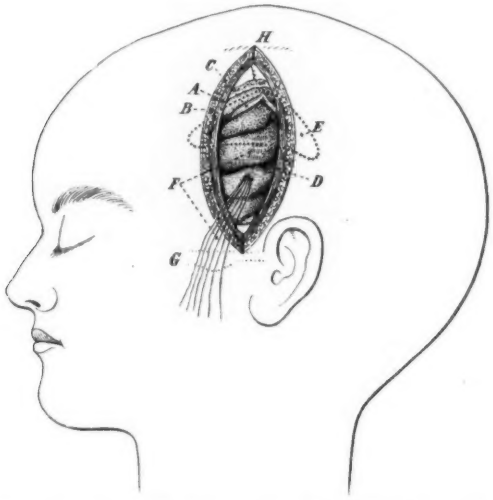

FIG. 1.—Diagrammatic sketch showing, through retracted temporal muscle (*A*), temporal fascia (*B*), and opened dura (*C*), the underlying cerebral convolutions and the Sylvian fissure (*D*). The dotted triangle (*E*) represents the lateral ventricle with the six linen strands (*F*) extending out from the ventricle through the upper temporal convolution beneath the Sylvain fissure. The scalp incision extends from the zygomatic arch (*G*) to the parietal crest (*H*).

externa. This fact has been so frequently overlooked that I feel that many needless and most tremendous operations have been preformed for the condition of hydrocephalus interna, which was not present—merely a hydrocephalus externa.

If we exclude those cases of internal hydrocephalus due to a tumor or cyst formation in the posterior midbrain and subtentorially producing a direct pressure blockage of the ventricles it is my belief that practically all of the cases of hydrocephalus are the result of a diffuse condition—an original meningitis in its various forms; if its consequent adhesions and exudative thickenings are so situated

as to block the exit of the cerebrospinal fluid into the subarachnoid spaces either in the aqueduct of Sylvius or at the foramina of Majendie and Luschka then we shall have produced the internal type of hydrocephalus; however, if the resulting adhesions, thickenings, etc., are not so situated as to block the ventricles, an internal hydrocephalus naturally does not occur, but still we shall have formed the external type of hydrocephalus because the cerebrospinal fluid cannot escape from the subarachnoid spaces of the cerebrospinal canal through its veins, sinuses, possibly lymphatics, etc., on account of their thickened walls and blocked stomata of exit due to the former meningitis of varying degree. Naturally, no

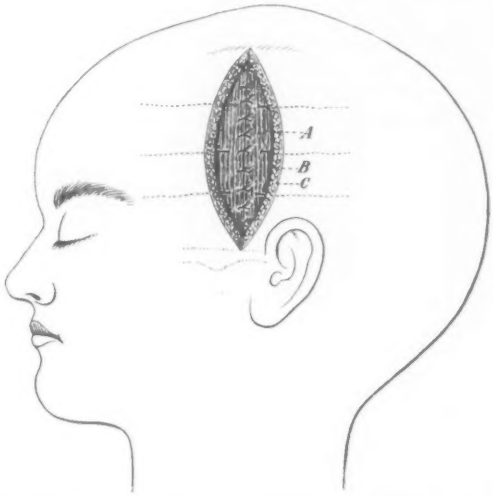

FIG. 2.—The six linen strands are brought out through the sutured temporal muscle (*A*) and temporal fascia (*B*) (to be sutured), and their ends are placed beneath the scalp (*C*) in the subcutaneous tissues—rich in lymphatics.

operative procedure merely designed to drain the ventricles into the subarachnoid spaces would be of any definite benefit in these cases; even if the openings of such drainage tubes did not themselves become blocked (and it is rare for them not to become blocked by adhesions, surrounding tissues, etc.), we should merely have the condition of internal hydrocephalus changed to one of external hydrocephalus, which is, to be sure, possibly a less serious condition in that the cerebral cortex is not so compressed and thinned as results from ventricular dilatation; besides, some cerebrospinal fluid may now escape through channels of exit only partially obstructed, and so a slight improvement is possible; however, unless the condition of external hydrocephalus is relieved so that the cerebrospinal fluid

can escape from the subarachnoid spaces and the cerebrospinal canal then the end result is merely delayed; a normal child is impossible.

It is interesting to note that epilepsy occurs more frequently in the external type of hydrocephalus than in the internal type; this may be due to the greater cortical irritation of this supracortical blanket of cerebrospinal fluid in these cases and also to the possibly greater corticomeningeal damage at the time of the original meningitis. Again, the great frequency of spina bifida associated with hydrocephalus merely illustrates the etiological factor in their production, a meningitic blockage of the stomata of exit in the walls of the veins, sinuses, possibly lymphatics, etc., so that the cerebrospinal fluid cannot escape from the spinal canal;[3] therefore an increased intradural pressure results in a protrusion and hernial sac formation in the lumbar region, the weakest and the latest region of the spinal closure; the fact that spina bifida occurs in about 35 per cent. of the cases of hydrocephalus, and that it is almost never associated with the internal type of hydrocephalus merely emphasizes the method of its production—that is, an early meningeal inflammation with a general blockage of the cerebrospinal fluid causing an increase of the intradural pressure so that the hernial protrusion of a spina bifida results with or without an external hydrocephalus; it is most rare, however, for a spina bifida to occur without there being definite signs of a mild external hydrocephalus.

Again, it has frequently been observed following the repair of a spina bifida, especially within a few days after birth, that a marked enlargement of the head occurred in a large number of the patients; this condition of external hydrocephalus might have resulted whether the spina bifida had been operated upon or not; but it does seem that the operative repair of the spina bifida by the removal of the large hernial sac, might have lessened the excretory surface of the cerebrospinal canal to such an extent that an increased amount of cerebrospinal fluid collected sufficient to cause the head to enlarge; it should be stated, also, that in these children the early operative removal of the spina bifida protrusion is usually performed when the hernial sac is enlarging, and thus the safety valve, as it were, of the increased intradural pressure, due to a blockage of the cerebrospinal fluid, is removed, and therefore the head, the next weakest portion of the cerebrospinal canal, will enlarge unless the fluid can be successfully drained.

In three patients I have been obliged to use the cranial method of drainage described in this paper after the spina bifida had been repaired. However, in this series of 41 patients an external hydrocephalus appeared several weeks after birth in 9 patients having the condition of spina bifida which had not been operated upon;

[3] Sharpe, Norman: Spina Bifida; an Experimental and Clinical Study, Ann. Surg., February, 1915, pp. 151–165.

in these cases it would seem that the spina bifida relief of the increased intradural pressure was not sufficient to prevent an enlargement of the head.

In this series of 41 operated patients we have used the same method of drainage, except in the first 2 cases, upon whom merely a bilateral subtemporal decompression was performed. Thirteen of the patients died, all of them with the exception of one child within thirty-six hours after the operation; that is, they could not survive the operative shock due to the sudden loss of a large amount of cerebrospinal fluid (this loss is now controlled and greatly lessened by elevating the head of the patient at the time of the ventricular opening). These patients were of the extreme type, very much emaciated, owing to their difficulty in swallowing, and were therefore bad operative risks. Of the remaining 28 patients upon whom the operation has been performed the results have been most encouraging. Their ages ranged from ten days to four years; naturally, the younger the child the less damaged is the cerebral cortex by the increased intracranial pressure; besides, the younger children stand the operative procedure as well and possibly better than the older ones, due most probably to the fact that many of the younger children are in a better physical condition than the ones subjected to a high intracranial pressure for a longer period of time; also to the fact that the cortical cells are not so highly developed in the younger children, and therefore they are apparently less upset by the operative procedure.

The object of the operation has been to drain permanently the ventricles (in the internal type of hydrocephalus) and the subarachnoid and subdural spaces (in the external type of hydrocephalus) outward beyond the cerebrospinal canal, that is, beyond the dura into the subcutaneous tissues of the scalp, a most absorptive area being well supplied with lymphatics. As drainage tubes buried in the body tissues always become blocked by adhesions, connective tissue, and surrounding tissues, etc., six linen strands are therefore inserted into the ventricles in the internal type and merely into the subarachnoid and subdural spaces in the external type of hydrocephalus and their ends brought out through the temporal muscle and temporal fascia beneath the scalp in a stellate manner; as these linen strands cannot be absorbed in the body tissues until four to six months have elapsed it is hoped that by the time of their absorption these artificial channels would be lined with endothelium or epithelium and thus their permanency and patency be assured.

In the laboratory the experimental production of internal hydrocephalus in puppies of ten days and two weeks of age has been performed by the method described by Dandy and Blackfan; that is, the aqueduct of Sylvius was blocked by a cotton plug through a suboccipital exposure; within twelve hours the ophthalmoscope

revealed choked disks of varying degree showing high intracranial pressure due to a blockage of the ventricles; unless this condition was relieved a secondary optic atrophy would result (as was permitted to occur in one dog so that it apparently became blind.)[4] In the other dogs which survived the first operation of ventricular blockage a subtemporal decompression was now performed (in three dogs a bilateral opening was made), and the linen strands inserted into the lateral ventricle and allowed to extend outward through the cortex, the opened dura, through the temporal muscle and temporal fascia into the subcutaneous tissues of the scalp. It was most interesting to observe a gradual subsidence of the choked disks in all but three of these nine dogs; in none of them, however, did the eye-grounds become as normal as before the operation, there being always an edematous blurring of the optic disks showing that the relief of the ventricular blockage had not been entirely complete. These dogs all died (three of them as a result of the internal hydrocephalus), within ten weeks after the drainage of the ventricles with the linen strands, which had therefore not yet been absorbed; so that it was still not ascertained whether the artificial canals formed by the linen strands would have remained as permanent patent channels of drainage for the cerebrospinal fluid or not.

However, one of the children having the condition of external hydrocephalus upon whom I had performed a right ventricular drainage on June 19, 1916, and then a left ventricular drainage on July 7, 1916, this child after making a definite improvement and three months after the first drainage operation, fell out of a high chair at home on September 9, 1916, fracturing the base of the skull. It was brought to the Polyclinic Hospital upon my service and died on October 13, 1916. An examination of the brain revealed an almost complete absorption of the linen strands used at the first operation, whereas the strands of the second operation were only slight absorbed. The report of the pathologist, Dr. F. M. Jeffries, regarding the condition of the artificial canals in the temporal muscle and fascia and in the subcutaneous tissues of the scalp showed that the linen strands brought out through the temporal muscle and temporal fascia at the first operation were almost entirely absorbed, being surrounded by numerous giant cells embedded in proliferative structures rich in bloodvessels and exudative cells. As this was a case of external hydrocephalus no linen strands had been inserted in the ventricles; it will therefore be necessary for us to wait until an examination of such a brain can be made—that is, a case of internal hydrocephalus—and at least six months after the linen strands have been placed in the ventricle in order to ascertain whether their artificial canals will remain as permanent patent channels of exit for the cerebrospinal fluid by

[4] This experimental work will be reported in detail later.

being lined with endothelial cells or not. The fact, however, that the children operated upon have improved and are still improving is of such significance to warrant the assumption that at least the artificial canals in the cerebral cortex are still patent.

With the exception of 6 of the total 28 children living after this operation of drainage, all of them have improved and are still improving; 5 of them have improved mentally and physically in every way, all of these children having been operated within one year after birth; 7 of the patients unable to walk before the operation are now walking, although with difficulty. The different stages of papillo-edema and choking of the optic disks producing a secondary optic atrophy and the visual impairment in these patients have been improved in each case unless a complete secondary optic atrophy had already occured.

Naturally, sufficient time has not yet elapsed to permit an opinion regarding the permanency of this improvement, and whether any of the younger patients will be normal and remain normal: but I do feel that the operative method here used does offer a chance of improvement to a large number of patients formerly considered hopeless; if a definite improvement can be obtained in the older children, then surely an earlier operation in the younger children when the condition is first observed and before the brain has been permanently damaged, should make a more hopeful prognosis possible. I am deeply indebted to Drs. McHenry and Hunt for their assistance; also to Dr. Lott for the apparatus devised by him to insert the linen strands into the ventricle; a description of it and the technic will be described in detail later.

EFFECT OF NUCLEIN INJECTION UPON THE LEUKOCYTES OF DOGS.

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and

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The result of work upon the effect of nuclein in experimental animals has not been uniform, probably because under this name both the acid and its sodium salt have been employed and no standardization has been attempted. In approaching this subject, we determined to employ the product accepted by the American

Medical Association, nucleinic acid or its sodium salt, and to use the phosphorus percentage as our guide.

In counting blood cells three pipettes were prepared for each count, three chambers counted of each pipette, and the number of leukocytes calculated from the average. Differential counts were made of 400 cells in each case.

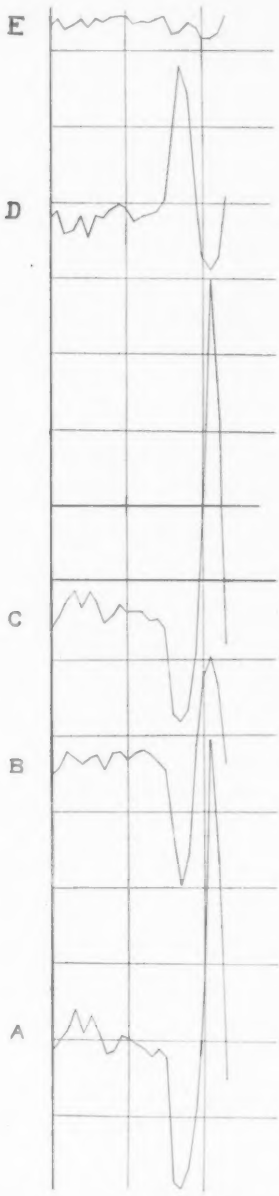
Counts were made immediately before the injection, and after the lapse of ½ hour, 1 hour, 2 hours, 4 hours, 7½ hours, 24 hours, 48 hours, 72 hours, and in some cases 96 hours after the injection. The effect of repeated puncturing of the dog's nose was controlled by taking counts on several untreated dogs. The effect of the injection of the menstruum in which the nuclein was to be administered was controlled by making counts after the intravenous and subcutaneous injection of physiological salt solution and 15 per cent. alcohol. The sodium nucleinate solution was prepared in 15 per cent. alcohol and physiological saline, in every case immediately before injection, and an amount of nuclein equivalent to 0.5 mg. phosphorus per kilo of dog weight was injected subcutaneously and intravenously in solution, 1 c.c. of which was equivalent to 1 mg. of phosphorus. The dog with which most work was done was a female of the fox terrier type, varying in weight from 7 kilos to 10 kilos in five months; of 24 counts taken in that time without treatment, the lowest was 7000 and the highest 13,000.

The injection of alcohol or of physiological salt solution, either intravenously or subcutaneously, was followed by no variation that did not fall within normal limits.

After the injection of sodium nucleinate, whether in 15 per cent. alcohol or physiological salt solution, whether subcutaneously or intravenously, there is a primary fall in the whole number of peripherally circulating leukocytes. This reaches its lowest point between the first and second hour, after which it starts to rise. The count is back to normal between four and seven hours, but the rise continues, until at the end of twenty-four hours the highest point is reached. From the twenty-fourth hour there is a gradual decline, the normal being reached between the seventy-second and ninety-sixth hour. In every experiment the curve greatly exceeded any figure for a similar period without treatment. The results of intravenous and subcutaneous injections are comparable, the clearness of the results, however, being greater by the former method.

Renner was able to raise the leukocytes 452 per cent. in his highest human case. Our greatest increase in the dog has been 290 per cent. of the original count.

The differential counts show after nuclein injection a relative and absolute decrease of polymorphonuclear neutrophiles, followed by a rise. This fall and rise of the polynuclear is largely responsible for the fall and rise of the whole count. Accompanying this curve of the polynuclears there is a similar downward and upward course



The curves are drawn to scale. *A*, variations in total number of white blood cells; *B*, variations in the per cent. of polymorphonuclear neutrophiles; *C*, variations in the number of polymorphonuclear neutrophiles; *D*, variations in the per cent. of small lymphocytes; *E*, variations in the number of small lymphocytes.

of the small lymphocytes, but it is of much less marked degree and more variable in its percentage; so much so, indeed, that no definite curve can be platted which will be followed in every experiment. In every case, however, the percentage of lymphocytes rises as the percentage of the polynuclears falls, and falls as it rises. This in itself shows that the gross variations in the total count must be due to polynuclear fall and rise. We are not now prepared to discuss the variations of the other leukocytes.

In a dog with an orginal leukocytosis and an eosinophilia the effect of the nuclein was the same as in the case of dogs with a normal blood picture.

In order to see if daily or twice daily injections of nuclein in 15 per cent. alcohol subcutaneously in quantities equivalent to 0.1 mg. of phosphorus per kilo would increase the leukocytes continuously and maintain them at a high level, a dog was given this treatment. There was a trifling rise of the whole number and of the percentage (therefore also the absolute number) of polynuclears. There was no continued rise, and the increase was irregular. This line of experimentation will be pursued further.

The Arneth formula in dogs is about as follows: 18, 40, 30, 7, 3. The injection of alcohol and saline has no appreciable effect upon it. Following an injection of nuclein, especially when given into the vein, there is a great relative and numerical increase of Type 1, cells with a single nucleus, amounting in one case to 52 per cent. of the polynuclears, while Type 2 remains about as normal and Types 3, 4, and 5 decrease. The rise of Type 1 seems to reach a high point in four hours corresponding to the time at which the leukocytes are on the increase and remains during the period in which the polynuclears are rising. Types 3 and 4 experience a fall roughly corresponding to the rise of Type 1, and later, corresponding again to the fall of Type 1, show a distinct increase, this arriving about the time that the leukocytes have resumed their usual numbers.

Nucleated reds have been encountered very occasionally in normal blood. After the injection of nuclein subcutaneously they are slightly increased, but when the substance is given into the vein they are increased quite markedly, in one case rising to eight times the number before injection. This rise appears during the time the leukocytes are falling. From a few observations it does not seem that there is any reduction in the number of red blood cells.

In 4 cases general toxic effects were noted after the intravenous injection of nuclein. The dogs became very restless for a few minutes, then lay quiet but trembling on the floor. After one and a half hours there was vomiting of a frothy yellow material, which vomiting was repeated throughout the day. On one occasion there were no general toxic symptoms.

Tentative conclusions from this work are that after nuclein injec-

tion there is a reduction in the circulating peripheral leukocytes, chiefly of the polymorphonuclears, and to a much less degree of the small lymphocytes, followed by a rise in both, but very largely of the polynuclears, even to 95 per cent. of the whole count. The total leukocyte count in dogs may be increased 290 per cent. in twenty-four hours. Repeated small doses of nuclein do not cause and maintain a high tide of leukocytosis. There seems to be destruction of polynuclears because of their great decrease, and the relative and absolute increase of young forms on the left side of Arneth's formula. The appearance of these young forms and of an increase in nucleated reds speaks for an increased activity of the bone marrow, therefore a stimulation of the tissue, but whether by the nuclein injected, as a response to the paucity of circulating white cells, or by excitation by the detritus of destroyed leukocytes we have not yet determined.

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THE LIMITS OF BLEEDING CONSIDERED FROM THE CLINICAL STANDPOINT.[1]

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There has been a tendency of late to overemphasize the importance of blood transfusion methods when in reality there is greater need for a more thorough understanding by the medical profession

[1] Read before the Semi-annual meeting of the Medico-chirurgical Faculty of Maryland, April 26, 1916.

at large of the indications for this procedure. It may well be that the cross-roads doctor is still unable to carry out a successful transfusion because of the complexity of present-day methods; one might even admit that the final word has not as yet been said on this matter; but the fact remains that for all practical purposes it is possible now for those properly qualified to do a successful transfusion with considerable ease, with absolute certainty and safety, on men, women, children, and infants, and by any one of several different methods. It goes without saying, of course, that to achieve the best results one must be prepared to use that method best suited to the case in hand, since no one device answers equally well in every instance.

If this is granted, then, it behooves the medical profession at large to learn what are the indications for this procedure and to become better acquainted as to the limits of bleeding, knowledge of which I have found to be sadly lacking. Indeed, it would have been strange had there been a wide-spread knowledge of this subject, since there was little to be gained by its intensive study prior to the advent of the practical transfusion. The therapy of anemia in its various grades has always been pretty well understood, and the treatment of actual hemorrhage from different causes has likewise been well recognized; so that heretofore it would have availed one little to study the actual limits of blood loss, with a view to relieving the condition when it got, so to speak, "out of bounds." The means of relief at one's command were insufficient. But times have changed. It is now possible to introduce fresh blood, literally at a moment's notice, into a circulation depleted by accident or disease. The only question to be determined is, When has the limit of bleeding been reached; and by limit of bleeding I refer to progressive anemia of any sort, from any cause.

The more I see of hemorrhage and anemia in general the more am I convinced of the utter futility of having a specific rule by which to be governed. Each case is a study unto itself, each individual represents an entity, each must be judged from all angles if one is to faithfully discharge his duties; and experience alone is the final great teacher in this as in all other phases of medicine. Yet one must have some tentative plans by which to be guided, and there are, after all, certain fundamental features which are true to a degree in all cases, and that are, if not regarded too literally, to be depended on. For instance, a sudden loss of blood is a much more serious matter than a gradual depletion. I once transfused a boy of about seven years who had had a progressive anemia of some months' standing of such grade that at the time of transfusion his hemoglobin was too low to register on any of the instruments commonly used for such work. Yet he was not in any immediate danger at the time of transfusion. And not long ago I transfused a woman whose hemoglobin actually registered 40 at the time of operation—

a reading that, under ordinary circumstances, is well within the limits of safety—yet this patient was in such a precarious state from sudden loss of blood, consequent upon a volcanic gastric ulcer, that the transfusion was terribly hurried.

A rapidly falling blood-pressure is always a warning of value, although it must be remembered that nausea of the slightest degree will send the pressure down. Pallor, the clammy, sweaty skin, the anxious countenance, are all danger signals that occur to you at once, and air-hunger when present is always of true diagnostic import.

In cases of severe hemorrhage—it matters not what the cause—a good working rule is to transfuse if the blood-pressure falls as low as 70 mm. of mercury, since life is hardly possible with anything below this level. But even in these cases the rule must be somewhat elastic because one can never tell when the pressure is going to rise. In some instances if the physician or surgeon in charge of the case has not taken the steps usual in emergency cases, such as salt infusions, etc., it may be wise to delay until these can be instituted, preparations for transfusion being made in the interval. If no appreciable results occur within one hour the case is usually hopeless unless new blood is introduced, and procrastination at this stage of the game is a fearfully dangerous plan, as the following story illustrates: Not many months ago I was called to see a woman who had had a placenta previa, and had been delivered in the manner usual for such conditions, a fair amount of blood being lost, though not nearly enough, according to the obstetrician in charge, to have caused the profound shock that ensued. The usual measures to combat such a condition were at once instituted and had been carried out before I reached the hospital. When I first saw her the patient was in a semiconscious condition, breathing was very shallow, and her pressure was around 70. Those in charge of the case, however, felt that in the fifteen minutes prior to my coming there had been a slight improvement, and the blood-pressure readings appeared to corroborate this, so that it seemed as if the condition might be one of pure shock, and that, if let alone, the patient might recover without transfusion. We waited just a half hour, but during those thirty minutes we lost our only chance, for suddenly the pressure dropped still further, the patient became absolutely unconscious, and died just as the transfusion was started. It is impossible for me to express the disgust I felt for my own judgment and my chagrin at the loss of that case. After years of study and after an experience varied and unique in hemorrhagic conditions I had begun to believe that I could really size up the situation in such a case. And the worst of it was that I had formulated this arbitrary rule of transfusion at 70 mm. pressure over two years prior to this case, and had never before broken it. Nowadays, I not only adhere to this rule religiously but go even a step farther—

when in doubt, my advice is to transfuse. It not infrequently happens that the pressure has not fallen to 70 mm., and never does reach that level, but the other features of the case are such as to render delay hazardous. Only once in my experience has a case been lost by transfusion; three have now definitely been lost by not transfusing, and in all three the opportunity was offered but not grasped. In fairness to myself, however, I will say that I advised against delay in two out of the three. In the case lost by transfusion the emergency was so great that time was lacking to carry out the usual hemolytic tests, and, as luck had it, the blood used was incompatible, hemolysis occurred, and the patient succumbed one week after the operation. This is the only instance of fatal hemolysis in my entire experience.

Curiously enough, the blood picture itself is of little avail in severe accidental hemorrhage. I once transfused a woman who was apparently exsanguinated from a gastric hemorrhage, yet her hemoglobin was around 50 per cent., and her red cells were over 2,000,000—figures well within the limits of safety. Even in cases of actual air-hunger, blood counts alone do not indicate the dire need for fresh blood. In fact, so constantly has this state of affairs been found in cases of this sort that this feature is now entirely ignored—I do not even go to the trouble to determine the hemoglobin or the red count. The explanation of this seeming paradox is that a sudden, terrific loss of blood apparently gives rise to a tightening up at first of the vascular apparatus, a narrowing of the vessel lumen, thus causing a concentration of the blood remaining in the peripheral system and at the same time preserving a blood-pressure sufficient to sustain life. The true anemia does not become apparent until later on, when the vessels have relaxed and taken up a renewed supply of plasma with the resultant blood dilution. It is, therefore, not infrequently an extremely difficult matter to determine the actual limits of bleeding. Cases must be judged in the aggregate, after a careful consideration of all features, and even then a positive answer is not always forthcoming.

I am not clear in my own mind as to the proper definition of so-called "shock," and it appears that many others have a similar hazy conception; but there is, without doubt, a certain something, commonly called shock, which complicates many of these cases. Not once but dozens of times the story has been that a comparatively slight loss of blood occurred—stories cited by men whose honesty was not to be questioned, whose keenness of observation is well recognized—still the patient looked as if there was not a drop left in the body, and blood-pressure readings were difficult to obtain because of a fluttering pulse. One such case followed a hip-joint manipulation by the open method; a fat embolus did not explain the condition found nor did the anesthesia, yet the patient died before transfusion could be instituted. Another

instance followed a dilatation of the cervix and removal of a child by version, giving rise to a fearful condition not explained by loss of blood alone. Prompt introduction of blood apparently righted the lost equilibrium. Is this condition due to anemia of the brain, or is it really connected with Crile's kinetic theory? I confess it to be beyond my ken. Having constantly to reckon with this phase of hemorrhagic conditions—for shock is nearly always associated with bleeding in the cases I am called upon to judge—I find it at the same time enigmatical and terrifying, for it introduces an unknown factor into conditions trying enough without it.

But before dismissing the subject of accidental blood loss, a few words might be in order concerning the use and abuse of drugs and salt solution in the treatment of these conditions. It is an awful thing to sit still and watch a man bleed to death, without so much as a decent attempt to save him. We all know that. It upsets everyone, makes everybody nervous, and this surely is the explanation of the hurry and bustle of an operating room in which the unexpected has happened, the delivery room in which the bleeding has been too great. But we might as well realize the truth; perhaps a proper realization will bring us to our senses. Drugs are of little aid in acute bleeding, morphin, judiciously given, excepted. Never have I seen the slightest benefit from strychnin, nor have nitroglycerin nor atropin been of much service. Digitalis and strophanthin may possibly momentarily support a faltering heart, but it is not the heart that is at fault. In the severe obstetrical bleedings that I have seen, even ergot and pituitrin have been of doubtful value. Yet the practice of giving one drug after the other, hypodermic on top of hypodermic, is still in vogue—so much so that it would be astonishing did I but enumerate the various "stimulants" given to one rather recent case. It is human nature to try to help, but drug therapy in acute bleeding is misdirected aid.

Personally, I think a quarter of a grain of morphin seems to be indicated at the start of any hemorrhage to quiet any restlessness; after that the doses had better be smaller, because of the depressant effect of this drug on respiration and blood-pressure. Measures to stop the bleeding should be instituted immediately, and the body should be kept as warm as possible, the foot of the bed elevated, the limbs bandaged, the patient kept quiet, and water given *ad libitum*. And by water, I mean liquids of any and all sorts, by mouth, per rectum, subcutaneous infusion, intravenously, coffee, tea, water, salt solution, ice—anything at all that will quench the intolerable thirst and keep up the bulk of the circulating medium—all within reason. And right here, perhaps, I may be permitted to sound a note of warning—too much has been expected of salt solution—far too much. The custom of giving salt solution (or water) per rectum after an operation of any magnitude is a good one, and the salt infusion in cases of bleeding is also good, as is at times

intravenous salt—all within reason. But it is ridiculous in the extreme to keep filling a patient full of salt solution just because a great quantity of blood has been lost. Salt solution will not turn into blood—yet it is all but expected to do so.

If I have seen one I have seen a dozen patients actually water-logged by salt solution—and with no improvement whatsoever. It never seems to occur to some men that a heart can be over-distended, that the blood can be made too dilute, that if 1000 c.c. or 1500 c.c. of salt do no good, a greater amount will be equally valueless. But time and time again have I seen infusions repeated after 2000 c.c. of salt have been given without any benefit at all. Everyone knows it is proper to give salt solution in cases of blood loss, but very few stop to consider how much ought to be given. Some three years ago I was asked to see a gentleman, aged sixty-five years, who had had a suprapubic prostatectomy done. He went off the table in very good shape, but a few hours later began to bleed, and had to be repacked. Even this did not stop the ooze. So about four or five hours after the operation he was taken to the operating room and thoroughly packed under light gas anesthesia. Rather shocked from this procedure, but still in fair shape, he was returned to his bed and readily took up 2000 c.c. of salt solution. In spite of this he gradually became weaker, and finally developed a Cheyne-Stokes form of respiration. When I saw him his blood-pressure was around 70 and he was in a semiconscious condition. Those in charge wished to delay because he was taking up the salt so well, but gave in after a brief argument. On incising his arm for a direct transfusion, not a drop of blood flowed from his tissues, but salt solution flowed very freely. The introduction of a few hundred cubic centimeters of blood from his son saved his life; and curiously enough the Cheyne-Stokes form of respiration disappeared while the blood was actually flowing into him.

It should be generally understood that if the bleeding has not been too great a few hundred cubic centimeters of salt are all that is needed to tide a patient over. In cases of very severe hemorrhage the amount might be increased a bit, but if 1200 c.c. do not steady a falling blood-pressure or cause a slight rise its introduction had better be discontinued. Even when there has been a rise the greatest caution must be exercised, for be it remembered that in these desperate conditions salt will frequently cause a rise in blood-pressure, but will not sustain it. When the bleeding has been excessive a transfusion is indicated, because it has been conclusively shown that blood alone can raise a pressure and sustain it. Salt solution has no sustaining power *per se*, and when the fall comes after a rise from this means it usually portends the end, for added salt solution is useless. It never raises a pressure twice!

One would think that little need be said concerning the limits of bleeding in the more gradual forms of blood loss, but experience

teaches otherwise. Not many weeks ago, toward 5 o'clock one afternoon, I was asked to see a little girl with a view to possible transfusion. On arriving at the hospital, and before going up to see the patient, the story was told that a slow nasal bleeding had been in progress for six weeks or more and that all measures of relief had been utterly futile. Her condition had not seemed alarming until some twenty-four hours previous, when signs of collapse appeared, which were combated by a salt infusion. As no permanent relief was afforded by this means, transfusion of blood was thought of, but when I saw the patient it was apparent that a transfusion was not for her—she was actually dying! Nor would I set these cases down as instances of sheer stupidity or ignorance, because the physicians responsible were well up in their general work and desired to do all in their power for their patients. The unfortunate feature was that, being unaccustomed to deal with intractable bleedings, they were uniformed as to the limits beyond which one dare not go without introducing fresh blood. It is quite possible, too, that some of them did not know that intravenous whole blood will almost always cause a cessation of a gradual ooze from most any cause—they failed to take advantage of their opportunities and their patients paid the penalty.

It should be understood that in cases of chronic bleeding the coagulation apparatus tends to become deranged, and the longer the seepage continues the less liable is spontaneous cessation to occur. And the curious feature is that in many of the worst of these cases, the coagulation time of the blood outside the body is but little delayed. I have no explanation for this phenomenon, but can vouch for its accuracy by repeated tests. Therefore, in cases of gradual bleeding from any cause too much must not be left to nature. Depletions from sloughing fibroids should never be permitted to progress beyond the safe operative stage; yet I have seen it done! No case of obscure internal bleeding in this enlightened day should progress so far as to need a transfusion; yet I have seen it done! No case of actual hemophilia should be allowed to go so far that the patient is exsanguinated before resorting to blood introduction; yet I have seen that done! The slightest bleeding in the newborn infant should be a signal to prepare for a transfusion; but is this done? By no means. Only a few days ago I was requested to see a two-weeks-old babe that had had a slight vaginal bleeding a day or two after birth, a few purpural spots later on, and finally an umbilical hemorrhage that resulted in death just a few moments after it was brought to the hospital for transfusion!

Let me emphasize that bleeding is a phenomenon of the gravest import. Little does one realize the vast number of lives lost yearly from this cause, since no statistics have ever been compiled on the subject. I had no idea of its consequences until some time after starting in transfusion work, but as the years roll by my list of cases

designated by the fateful words "too late" grows perceptibly, until now I take it as a matter of course that not an inconsiderable percentage of the cases I am called to see are beyond help simply because of an insufficient knowledge concerning the limits of bleeding. In fact, it was a study of these "too late" cases that prompted me to take up the subject in the hope that a renewed interest might be awakened.

Even the chronic anemias are, from my view-point, permitted to progress entirely too far. I ask why is it necessary to delay transfusion in cases of pernicious anemia until the victim has a hemoglobin so low that an accurate reading is extremely difficult —15, 12, even 10 per cent.—and the patient is pale as death and so weak that transfusion is an extremely dangerous undertaking? Fresh blood does not cure these cases, that I well know, but in general it will cause a remission after all other measures have failed. Then why not transfuse, if transfuse at all, before the very bottom has been reached? Some may say that one can never tell when drug therapy will start the rise? If so, I agree with them, but in my experience after drugs have caused one or two remissions a continuously falling hemoglobin under this therapy is a danger signal, and when the hemoglobin reaches say 30 per cent., why not introduce new blood and give the drugs a bit more to work on?

The same thing may be said of the indefinite undiagnosed anemias, some of which are permanently cured by transfusion. And the intestinal bleedings from ulcers, duodenal, gastric, may be placed in the same category. The convalescence of many of these patients could be materially shortened by the introduction of whole blood. Why compel them to start from the depths and rebuild entirely when one knows it to be so difficult and tedious when, by repeated demands, the blood-forming organs have been strained to the breaking point? And since many of these individuals have difficulty in getting beyond a certain point in their rebuilding process, it has often occurred to me that the impetus resulting from a medium-sized transfusion at this point might be a stimulus of inestimable value in healing the ulcer and finally regaining complete strength and health, a point that has hitherto had but passing consideration.

And how often are surgeons called upon to operate on patients whose strength has been sapped by some chronic wasting process that is only another form of bleeding? Deaths from such operations are not infrequent, yet poor surgical risks of this sort can be turned into good operative risks without the slightest difficulty by a transfusion prior to operation, or, as I have frequently done, during actual operation. One case in particular, a little out of the ordinary perhaps, but extremely instructive, may be cited: A man came in suffering with a double pus kidney. He stood one operation fairly well, but failed to respond after drainage of the other side. For days all measures of relief were tried, but he continued to slip away,

and his last kidney wound became a foul, sloughing cesspool, while his temperature was continually mounting. A blood transfusion changed this individual from one absolutely lost to a man of hope; his temperature dropped, the wound in his side gradually cleared, healthy granulations sprang up, and he left the hospital in good shape not many weeks later. And I maintain that is a triumph of the most fundamental character.

In conclusion, I would urge that henceforth transfusion be not regarded entirely as a measure of last resort—food for the reporters by spectacular stories of the dead brought back to life. A more thoughtful attitude concerning blood loss from any cause is greatly to be desired, and the phenomena accompanying bleedings of all magnitudes should be carefully noted and studied, with the view to improving the general knowledge of such matters. Furthermore, a partial revision of the existing ideas of combating hemorrhage and the condition commonly known as shock may possibly be of material advantage in view of the fact that transfusion may be done so readily at the present time. But above all, surgeons and physicians should learn to recognize the limits of bleeding and to act promptly. In the acute hemorrhages, according to my experience, the safest guide is the blood-pressure; in the chronic bleedings and the anemias it is the hemoglobin; but one must never lose sight of the fact that at times all signs fail, and there remains naught for guidance but experience and judgment. Under these circumstances my advice is, "When in doubt, transfuse!"

THE MAIN FACTORS AFFECTING THE INTENSITY OF SOUNDS AS THEY PASS FROM THE INTERIOR OF THE LUNGS TO THE PERIPHERY OF THE CHEST.

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The intensity of sound as it passes between two places may vary in one of three ways: It may become intensified, it may remain constant, or it may be diminished. In these general features sound presents certain resemblances to light. Factors operating to increase sounds between the interior of the bronchi and the periphery of the chest are little understood and are probably of very minor importance, as far as the mere intensity of audible sounds is concerned. Factors concerned in maintaining the original intensity of sounds are found particularly within the bronchi, which act much

as speaking tubes in preventing, through limitation of diffusion, the weakening of the sounds coming from the upper respiratory tract, while at the same time the bronchi carry the sounds deeply within the lungs, thus preserving a good deal of their original intensity. The bronchi, however, provided they are patulous, and not too far retracted toward the root of the lung, exert their influences on the sounds within them under both normal and pathological conditions, so that to a large extent our interest centres on the sounds *after they have escaped from within the bronchi.* It is not, however, with an increase in sounds, or their retention, that we are chiefly concerned, but with those factors tending to effect a diminution in the intensity of sounds in the chest.

THE DIMINUTION OF SOUNDS. The diminution of sounds in different clinical conditions as they pass from the interior of the bronchi to the periphery of the chest, depends chiefly on one or both of two factors: *the diffusion of sounds* and *the reflection of sounds.*

The Diffusion of Sounds. By diffusion is meant a spreading out of the sound, a dilution of the sound energy. If a sound originates at a point, and, owing to the non-existence of any obstructions, spreads out equally in all directions, the intensity of the sound will be the same on all points of the surface of a sphere whose centre is at the point of origin of the sound. At the surface of a second sphere, double the area of the first, and concentric to the same point of sound origin, the vibration energy, per unit of area, and hence the intensity, is halved. This is a deduction from the so-called inverse square law of acoustics, which tells us that, in a medium in which the absorption of energy is small enough to be neglected, and which is extended enough (infinite) so that the complications introduced by boundaries may be disregarded, the intensity of a sound varies inversely as the square of its distance from the source. The importance of the factor of diffusion of sound in diminishing the intensity of sounds as they approach the surface of the chest has, for the most part, been understood in only a very general way, and the great relative importance of this factor in certain clinical conditions has been largely overlooked. Thus, the main factor in determining the diminution of vocal resonance in pleural effusion as contrasted with the normal lung, is diffusion.

The Reflection of Sounds. The reflection of sounds is a form of sound obstruction, and is as different from the diffusion of sound as the reflection of light is from the diffusion of light. Of course we are now speaking of reflection as it interferes with the transmission of sounds on their way to the hearer who is situated distal to the point of reflection, for reflection may intensify sounds by actually focussing them to a smaller area. In the chest we look for factors reducing sounds (exclusive of diffusion) either within the different individual media encountered, or at the junction of different media, that is, where different media occur in combination.

THE DIFFERENT INDIVIDUAL MEDIA CONCERNED. All the individual media with which we are concerned may be divided into three groups: (1) the solids, or, better, the gels, (2) the fluids, and (3) the gases. It is very easy to show that *each of these individual media by itself offers but little obstruction to the passage of sound through it, and that whatever proportion of sound is lost in its passage through these individual media is due to diffusion.* In the de-intesification of sounds on their way to the periphery of the chest, no differences in the conductive properties of any of these media, solids (or gels), fluids and gases, have been determined by the stethoscope, and therefore are not of present concern to us.

Different Media in Combination. Apart from diffusion the chief factor in diminishing sounds passing through the chest is the reflection of sounds occurring at the junction of certain of the above mentioned media. *Comparatively little sound is lost in its passage between fluid and tissue (or solid), because their densities are not greatly different and they are both highly elastic. But between air and tissue or air and fluid, provided the body of tissue or fluid is not too attenuated, a very marked "break" occurs, because of the vast differences in density of the adjoining media.* As far, then, as different media in the chest affect sound intensity by their combination, they may be roughly reduced to two: a medium of air and a medium free from air.

To summarize what has been said about the diminution of sound in the chest, we are dealing chiefly with two factors—diffusion and reflection—the former occurring fairly constantly, the latter for the most part requiring the presence of air and taking place mainly at the junction of air with either solid (tissue, gel), or fluid. Or, to put it in another way, there is very little diminution of sound in the absence of air, except that due to diffusion, provided the bronchi are patulous and not reduced in size.

In speaking of the combination of tissue and air as offering an obstruction to the passage of sounds, the degree of obstruction will depend on the thickness of the tissue adjacent to the air. Thus, much more sound is lost when the vibrations are passing from air to chest wall, than from air in one of the vesicles in the lung to the delicate enveloping membranous wall. The fact that sounds pass though the normal lung with as little loss as they do, is largely because each delicate membrane individually offers relatively very little obstruction. *More obstruction occurs when these membranes are tense than when relaxed, the greater tenseness of the membranes in the former case effecting a greater degree of reflection of the vibrations.* The result is the well known clinical fact, that the normal lung in the normally distended state impedes the transmission of sound more than when in a state of collapse. *A great difference in transmissability can be shown to exist when the sounds traverse equal distances in distended and relaxed lungs.*

While the solid tissues of the lung (normal and diseased), and of

the chest wall in the living body contain a large proportion of fluid in the form of lymph and blood, this mixture of two different media, fluid and tissue, does not materially reflect or obstruct the passage of sounds, for the reason given, that the densities of tissue (normal and pathological) and fluid are relatively alike.

The purely solid portions of the tissues we are considering are made up chiefly of soft cellular substance, which, to a large extent at least, is physically allied to gelatin, and belongs to the group of gels, substances which it is well to bear in mind are very different physically from such solid materials as wood and metal. These gels, though potentially capable of yielding a large proportion of fluid, do not actually contain it, just as gelatin, though capable of yielding much water, does not do so as long as it is in the state of gelatin.

Absorption, Resonance and Interference of Sounds. Besides the main factors of diffusion and reflection of sounds there are other factors affecting the intensity of sounds within the chest, but their activities, what little we know about them, appear to be much less influential. On the quality of sounds, however, at least in the case of resonance, their effect may be very marked. By *absorption* is meant the annihilation of sound energy as such, and its conversion into other forms of energy, such as heat, which may not be appreciated by the ear. It is a factor concerned in the diminution of sound intensity quite distinct from diffusion, though at times confused with it. Absorption by different media apparently plays a very small part in the reduction of sound intensity in the chest. *Resonance* acts particularly by affecting sounds of a certain pitch, and as the chest sounds familiar to us are compounded of many different kinds of individual sounds, resonance usually exerts its influence only on a limited number of these individual sounds. Certain elements in the original sound may be diminished through *interference*, that is, the superimposition of sound waves upon each other.

The Size of the Bronchi as a Factor in Determining the Intensity of the Vocal Resonance. When a bronchus, or a group of bronchi, such as we find in the bronchial tree of one lung, become reduced in size, the total amount of sound energy coming from the upper respiratory tract and being admitted to these bronchi, is less than when the bronchi possessed their original larger capacity. How much of an effect this decrease in the bronchial capacity may exert upon the vocal resonance requires further investigation. This factor would have a particularly favorable opportunity to operate when the whole lung is much reduced in size, as may occur in pleural effusion or pneumothorax. Braune and Stahel[1] have shown that

[1] Ueber des Verhältniss der Lungen, als zu ventilirender Lufträume, zu den Bronchien, als luftzuleitende Röhren, Arch. f. Anat. u. Physiol., 1886, Anat. Abtheilung, S. 5.

the diameter of a bronchus usually depends in part upon the amount of air in the lung, and it seems quite possible that in chronic affections of the lung and pleura the bronchial capacity might become sufficiently reduced to appreciably affect the vocal resonance. The explanation for the greater vocal resonance often encountered normally over the right lung, may rest partly on the fact that the right lung is larger than the left, which necessitates a larger total intrabronchial capacity on the right side, which permits the entrance of a larger amount of sound energy into this side. (The ratio of the diameters of the right and left main bronchi in the normal individual, according to Braune and Stahel, is about 4 to 3, approximately the same ratio that they obtained for the respective weights of the lungs on the corresponding sides.)

The Origin of the Sounds Involved. It is to be recalled that the spoken voice sounds, bronchial breathing, and whispering pectoriloquy arise in the upper respiratory tract, so that the source of these sounds is identical, however variable may be the conditions in the lungs, and different intensities in different parts of the external chest surface must be due to factors operating distal to the source of these sounds. On the other hand, variations in intensity of the vesicular breath sounds, at least of the inspiratory ones, sounds which arise within the lungs, may be due in whole or in part to differences in intensity at the site of production of these sounds. Thus, in pleural effusion, even if slight, the vesicular murmur may be weakened because there is not proper expansion of the vesicular structure to produce sounds of normal intensity.

The Relative Importance of Different Portions of the Respiratory Passages below the Larynx as Direct Sources of the Vocal Resonance. While the trachea and bronchi resemble speaking tubes to the extent that they transmit within their interior sounds coming from the upper part of the respiratory tract toward peripheral portions of the lungs, the walls of the tracheobronchial tree are, all along the route, taking up a certain amount of the sound present within these air passages. Some of the vibrations entering these walls are never perceived by the auscultator, and, in the case of the normal lung, the vocal resonance over most of the chest is due entirely or largely to vibrations escaping from the smaller bronchi. Under these conditions the trachea and larger bronchi serve only as carriers of sound from the upper respiratory tract to the smaller bronchi. The normal right apex, according to Fetterolf,[2] receives some audible vibrations that have come directly from the trachea without having had to pass through the bronchi. In pneumonia it has been shown by Mason[3] that the voice

[2] The Anatomic Explanation of the Greater Amount of Vocal Fremitus and Vocal Resonance Normally Found at the Apex of the right Lung, Archives of Internal Medicine, February, 1909.

[3] American Journal of Diseases of Children, March, 1916.

and breath sounds become much altered when a peripherally situated consolidation, previously separated from the large bronchi by an area of normal lung tissue, by extension centrally, finally comes in direct contact with these large bronchi. The large bronchi furnish some of the sound when the lung assumes the fetal type following the disappearance of air from within the alveoli. The trachea itself may be a direct source of sound when the upper part of the lung is solid, or when the upper portion of the pleural cavity is filled with air or fluid, the pulmonary apex being free from adhesions and retracted toward the root of the lung.

While fully recognizing the limitations of the explanations offered for various auscultatory findings obtained over the lungs, we believe that the application of the simple principles just presented to certain clinical conditions can frequently be determined to a considerable

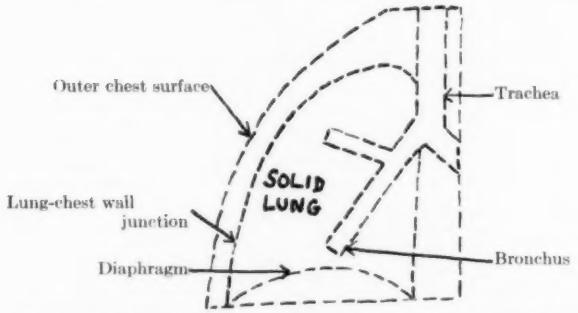

FIG. 1.—Solid lung. No special reflection, the entire distance between bronchial wall and outer chest surface. Diffusion an important but not a special factor. More sound retained within the bronchi, at least, the small ones, than normally. Vocal resonance increased. Bronchi in diagram represent small as well as large bronchi.

extent. Six of these conditions will receive brief attention: solidified lung, normal lung in a state of normal distention, collapsed but otherwise normal lung, pleural effusion with diminished vocal resonance, pleural effusion with increased vocal resonance, and pneumothorax.

Solid Lung. This is the simplest clinical condition we have to study. In tracing the sounds from within the bronchi through the solid lung to the periphery of the chest we find the factor of diffusion of sound everywhere operative. The activities of this factor are very much limited within the bronchi because the bronchial walls to a great extent reflect the vibrations back into the interior of the bronchi, this reflective action in the smaller bronchi being even more powerful when the lung is solid than when it is normal. Once the vibrations have entered the bronchial walls, however, their further course is pursued entirely through tissue with

its associated fluids, the solid lung and chest wall forming practically a single good conducting medium which offers very little obstruction to the passage of the vibrations, so that *nearly all the sound intensity that is lost en route from bronchus to chest periphery is due to diffusion.* In no other common clinical condition do we find so little tendency to the reduction of sounds from reflection as when dealing with a solid lung. And even the degree of diffusion is not greater than that ordinarily encountered. The voice sounds are, therefore, relatively loud, or, as we usually express it, are increased in intensity over a solid lung. In addition the weaker sounds produced in the upper respiratory tract, bronchial breathing and whispering pectoriloquy, become audible at the chest surface, while the normal breath sounds disappear because they are not produced.

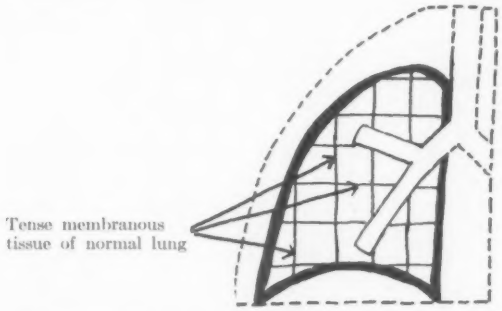

FIG. 2.—Normal lung. Special reflection (indicated by continuous lines), first, at junction of the air in the lung with the chest wall; second, at junction of tense membranous tissues of lung with air in the vesicles, and, possibly to some extent; third, at junction of bronchi with air in adjacent vesicles. Diffusion much as in the case of solid lung. Vocal resonance normal. Bronchi in diagram represent small as well as large bronchi.

The Normal Lung Normally Distended. The conditions present in a chest containing normal lung in its normal state of tension offer a marked contrast to those occurring when the lung becomes solidified. Such a lung, unlike the solid lung, does not exhibit a single good conducting medium free from air between bronchial wall and chest wall. Instead, we find air associated in a very peculiar way with the delicate membranous tissues of the pulmonary parenchyma, these membranes at the same time being under a considerable degree of tension. Sound energy is lost, (1) as the vibrations pass from the bronchial walls to the air in the nearest adjoining vesicles; (2) as the vibrations pass between the air in the vesicles and the tense membranous walls enclosing them, and (3) as the vibrations pass from the air in the vesicles immediately underlying the chest wall to the tissues between these vesicles and

the chest wall, which practically means to the chest wall. *Each such loss of energy is the result of reflection* acting on the vibrations as they pass toward the exterior of the chest. It is not surprising, therefore, with all these places unfavorable to the transmission of sound, that the normal vocal resonance is so much less intense than when the lung is solid. In fact, it is more difficult to explain why the voice sounds are not actually weaker than they are. Two conditions may be mentioned which are relatively favorable to sound transmission when the lung is normal: (1) Sound can more easily escape from within the smaller bronchi to the thin walls of these smaller bronchial tubes of the normal lung than when these tubes become encased in solid tissue. (2) There is some sound conduction even in the thin membranous tissue of the normal lung. The absence of bronchial breathing and whispering pectoriloquy over normal lung is due to the same conditions that make the vocal resonance less than that over solid lung.

Relaxed but Otherwise Normal Lung. Clinically this condition occurs above a pleural effusion and is apparently responsible for the increased vocal resonance frequently found above an effusion. A good deal of the confusion and error that has resulted in the attempt to explain different degrees of intensity in the sounds heard over the chest has been due to a failure to recognize *the different effects on sound transmission occasioned by the normally distended lung and the collapsed lung*, or, as it is frequently termed, the compressed lung. Bullar (1884) was the first one to describe the marked differences in conductivity of these two kinds of lungs, though he offered no explanation for this variation. *The relaxed lung allows vibrations to pass through it with less obstruction or reflection than the distended lung*, for the reason given in speaking of the normally tense lung, that is, *because the relaxed membranes of the collapsed lung are not under tension*, and therefore reflect the vibrations backward to a much less extent than when the same membranes are tense. This principle is well known, and can be readily illustrated by passing sounds into a tube and listening at the far end, when it will be noticed that if a thin piece of rubber membrane (or body tissue) is placed across the lumen of the tube at some point, much less sound gets through to the far end of the tube when this membrane is held taut than when it is completely relaxed. The relaxed lung lets more sound through it than the distended lung even when the area traversed in the two cases is identical. In all respects, *except the degree of tension of the finer tissues*, the relaxed and tense lungs are very much alike as far as sound transmissibility is concerned, when equal distances in the two kinds of lungs are compared.

Pleural Effusion with Diminished or Absent Vocal Resonance. The explanations for this phenomenon when not entirely erroneous have been either obscure or incomplete. The abnormal

conditions encountered in a simple case are the presence of fluid within the pleural cavity, and a more or less relaxed lung. The clinical fact above referred to, that the vocal resonance above a pleural effusion is frequently increased, indicates that the sounds escape from the relaxed lung to adjacent tissue with more intensity than in the normal case. The evidence indicates that the sounds pass between a relaxed lung and fluid with about an equal intensity to that found in the passage of sounds between relaxed lung and tissue. Therefore, in those cases presenting an increase in vocal resonance above an effusion we conclude that the vibrations entering the fluid are louder than those entering the chest wall of an individual with the lung in a normal state. *Any loss of sound, then, (except as noted below), encountered over a pleural effusion, must take place after the vibrations have entered the fluid.* We have already seen

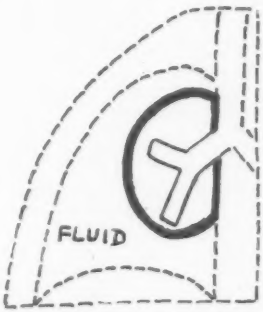

FIG. 3.—Pleural effusion. Lung air-bearing, but membranous tissue relaxed. Special reflection (indicated by heavy continuous line) at lung-fluid junction. Reflection at junction of bronchi with adjacent intravesicular air (indicated by light continuous lines) of questionable importance. Special diffusion between lung and chest wall. Vocal resonance decreased. Bronchi in diagram represent small as well as large bronchi.

that tissue alone, and fluid alone, and even the combination of the two, offer very little impediment to the passage of sounds. *The loss of sound encountered over an effusion may be traced largely to diffusion*, the result of the greater distance occurring between the lung and the chest wall overlying the fluid as compared to the close approximation of the lung to the chest wall under normal conditions when no fluid separates them. The sounds become weak over an effusion for the same reason that they do over the liver, because of diffusion and not because of reflection. When the lung is much reduced in size the diminished vocal resonance may be partly due to the decrease in size and capacity of the bronchi, which allows less sound to enter the lung than normally. When effusion is present the vesicular murmur may be diminished or absent partly because the vesicles are not expanding properly to produce sounds of normal intensity.

Pleural Effusion with Increased Vocal Resonance. The explanations offered to account for an increase in the vocal resonance sometimes met with over a pleural effusion are few and for the most part unsatisfactory. *In some cases this increased vocal resonance is apparently due to the fact that the fluid adjoins a lung which is solid.* The lung may be solid simply from absence of air in the vesicles, as when it assumes the fetal type after prolonged collapse, or it may be solid as a result of inflammatory or other products in the vesicles. It is important that no air should remain in the pulmonary parenchyma. *When a solid lung is separated from the chest wall by fluid there is very little reflection or other form of obstruction to the passage of vibrations on their way to the periphery of the chest, after they have entered the bronchial walls. Neither the tissues (lung and chest wall), nor the fluid, either individually or in*

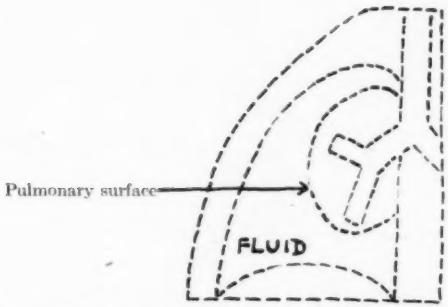

FIG. 4.—Pleural effusion. Lung solid. No special reflection, the entire distance between bronchial wall and outer chest surface. Compare solid lung. Special diffusion may exist between lung and chest wall. Vocal resonance may be increased. Bronchi in diagram represent small as well as large bronchi.

combination, offer any serious impediment to the passage of sounds. If, then, diffusion is limited, the conditions are very much as in the case of the solid lung unassociated with fluid. And whatever the degree of diffusion, due to separation of the lung from chest wall and other factors, the conditions are very much the same as though tissue replaced fluid in the pleural cavity. The explanation is entirely satisfactory where the solid lung is separated only a short distance from the chest wall by the fluid, but it is well known that very large effusions on either side of the chest separating the lung a great distance from the chest wall, and thereby greatly increasing the diffusion between lung and chest wall, may furnish striking examples of cases with increased vocal resonance. In such cases it seems likely that some vibrations are received at the periphery of the chest *coming straight across through the fluid from the trachea.* The physical conditions are certainly favorable for this,

that is, the fluid may be present in the upper part of the pleural cavity, so that in the course of the vibrations from the trachea to the chest wall no air intervenes, but only tissue and fluid. Very

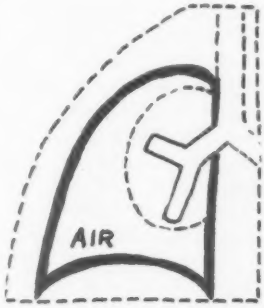

FIG. 5.—Pneumothorax. Lung air-bearing. Special reflection (indicated by heavy line) at air chest-wall junction. Reflection at outer bronchial surface as in Fig. 3. No special diffusion; diffusion being limited by the heavy walls enclosing the air in the pneumothorax cavity. Vocal resonance distinguished. Bronchi in diagram represent small as well as large bronchi.

loud sounds may be obtained over the chest of a fetal calf after fluid has been introduced between the chest wall and the solid fetal lung, these sounds becoming much weaker, however, after inflation of the lung. (The sound in this case is introduced into the trachea.)

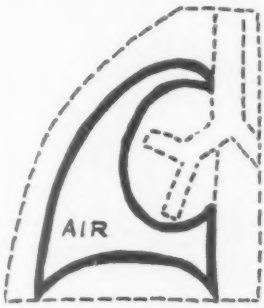

FIG. 6.—Pneumothorax. Lung solid. Same as Fig. 5, except that there is special reflection (indicated by heavy continuous line) between solid lung and surrounding air, while there is no reflection between outer surface of bronchi and surrounding solid tissue. Vocal resonance diminished. Bronchi in diagram represent small as well as large bronchi.

Pneumothorax. In the case of pneumothorax the distinctive location of sound loss is at *the junction of the air in the pneumothorax cavity with the chest wall,* where reflection of the vibrations

acts very powerfully as a result of the enormous differences in density of air and tissue. If the collapsed lung is air-bearing the opportunities are more favorable for the transmission of sounds to the periphery of the chest than if the lung is solid, because more sound can enter and leave a collapsed air-bearing lung under these conditions than when the lung is solid, while at the same time the air-bearing lung itself is a very good transmitter of sound. If no adhesions exist at the apex sound may be obtained coming directly from the trachea to the chest periphery by way of the pneumothorax cavity. *Diffusion exerts little effect between the lung and the chest wall*, the sound intensity being fairly equal throughout the pneumothorax chamber, because the air is enclosed within solid walls. The diminished vocal resonance is probably partly due to the small size of the collapsed lung and the consequent diminished calibre of its bronchi, resulting in the admission of a reduced amount of sound into the affected lung. When a patulous fistula exists between the pneumothorax cavity and a bronchus, the conditions are favorable for abnormally loud vocal resonance.[4]

[4] Some of the subjects briefly presented in this paper have been treated more fully in the Tenth Report of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis, in an article by Montgomery and Eckhardt, entitled "Pulmonary Acoustic Phenomena."

REVIEWS

DISEASES OF CHILDREN. By EDWIN E. GRAHAM, M.D., Professor of Diseases of Children, Jefferson Medical College, Philadelphia; Pediatrist to the Jefferson Hospital and to the Philadelphia Hospital; Consulting Pediatrist to the Training School for Feebleminded, Vineland, N. J.; Member of the American Pediatric Society, etc. Pp. 902; with 89 engravings and 4 plates. Philadelphia and New York: Lea & Febiger, Publishers, 1916.

THIS work makes another addition to the rapidly growing number of complete text-books on Pediatrics. In general it conforms to the best orthodox forms of such publications brought "down to date." Apart from a general commendation of a book which the practising physician may accept as giving a reliable account of disease in childhood, certain features may be especially noted. The chapter devoted to infant mortality, heredity and environment is most timely and appropriate and should indicate to every thinking physician the importance of "preventive" as contrasted with "curative" medicine. Another chapter on the role played by fresh air in the treatment of disease should also be included in the same category as it forms a plea for the value of fresh air in health as well as in sickness. A special chapter on puberty discusses in a much more extensive manner than is usual a subject upon which the average physician has ideas scarcely less hazy than those of the laity. Another chapter of practical importance is that on dentition—that much overrated but not to be overlooked unit in the physiological development of the child. The relation of the teeth to disease also is adequately considered. Throughout the book treatment is indicated in a way which is most likely to benefit the general practitioner, with the proper warning against the much-to-be-deprecated overzealous medication. The subject of infant feeding admittedly is the most difficult one to present in the whole range of pediatrics, and it would seem to the reviewer that the author has not developed it in the most logical order. The information, for the most part, is there, but the difficulty of securing an adequate conception of the relation of food to digestion and the results of indigestion has not been solved as satisfactorily as the author's knowledge and experience would warrant. Another point for criticism is to be found in the failure to follow the modern tendency toward the simplification

rather than the multiplication of diagnostic titles, especially on the subject of the digestive disorders of infancy. A few omissions are to be noted, but on the whole the book covers the range of the diseases of childhood in a manner more than usually thorough.

J. C. G.

A TEXT-BOOK OF HUMAN PHYSIOLOGY. By ALBERT P. BRUBAKER, A.M., M.D., Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College. Fifth edition. Revised and Enlarged; with 1 colored plate and 359 illustrations. Philadelphia: P. Blakiston's Son & Co.

AFTER paging through this new edition and comparing it with the previous edition one is impressed with the thoroughness of the revision. Changes are numerous but not violent. This indeed would have been a mistake for the preceding edition was not only pedagogically sound, but satisfactory as to the relative importance which was given to the data presented. The thoroughness rather manifests itself in the form of splendid conservatism in the selection of new material. In spite of the introduction of much new data the book is little enlarged, for many omissions of the proper sort have been made.

Changes of a minor character are seen in the first chapters on the chemical composition of the body, the physiology of the cell, of the skeleton, of the muscle tissue, and of the nerve tissue. Diagrams and illustrations have been modified, and sometimes replaced by others. Much more bold type is used, which make the volumes more efficient as a reference book. These changes distinctly improve these chapters which had already reached a high degree of excellence in the previous edition.

The chapter on food has undergone considerable change. The description of a modern metabolism experiment is concise and clear. The chapter on digestion shows considerable revision. The physiology of the sphincter cardiæ, and the movements of the intestines has been brought up to date. In the chapter on circulation a couple of pages are devoted to the electrocardiogram. Arterial pressure is also given more consideration. The physiology of the vasomotor mechanism also shows advantageous changes of treatment and some new material is added. Calorimetric measurements are considered in greater length in the chapter on respiration. Internal secretions have also received much attention, for changes of an important character are noted. If space permitted it could be shown that the other chapters have received painstaking attention as indicated by the changes of both a major and a minor character.

The book has been written by an author who knows the philosophy

of pedagogy and who is widely read in the various ramifications of the subject. He knows well how to weigh the relative importance of the material presented by researchers of the present day and to combine it with the older information in the proper proportion. E. L.

DISEASES OF THE DIGESTIVE TRACT AND THEIR TREATMENT. By A. EVERETT AUSTIN, A.M., M.D., Assistant Professor of Clinical Medicine, in Charge of Dietetics and Gastro-intestinal Diseases, Tufts College. Pp. 552; 85 illustrations. St. Louis: C. V. Mosby Company, 1916.

THIS volume, the latest arrival in the rapidly growing number of monographs dealing with gastro-intestinal subjects, while not so good as some of its predecessors, nevertheless presents features which speak in its favor.

While by no means an exhaustive treatise of the subject, and not intended as such, it contains a great deal of practical information, derived from a clinical experience covering a quarter of a century, which should prove useful to both students and general practitioners, although it may not appeal so strongly to men who are specializing in this subject and who are familiar with the best books in this and other languages.

To the reviewer, who does not wish to be unduly critical, it would seem that the book is badly balanced in regard to the space allotted to different topics. For instance, one-half of the volume is taken up with a discussion of the anatomy and physiology of the gastro-intestinal tract; the examination of the patient, historical, symptomatic, and physical; the methods of applying laboratory aids in diagnosis; a chapter on dietetics, which is one of the best in the book; and a chapter on treatment in general, which is fairly well presented and forms a good working basis, though unfortunately is far from being up to date. These chapters make up Part I of the volume. Part II is devoted to a discussion of special gastric diseases which, in the opinion of the reviewer, are afforded scantier treatment than they deserve, only 66 pages being given to the organic diseases and 24 pages to the functional disturbances. In regard to the former only five conditions are presented, namely, gastritis, ulcer, ectasia, cancer, and splanchnoptosis, no mention being made of sarcoma, syphilis, tuberculosis, benign tumors of the stomach, benign pyloric stenosis, and congenital defects, topics which while of less importance than those discussed, nevertheless deserve a place in a treatise of this size. Again, in regard to functional disturbances no mention is made of some of our more recently acquired knowledge of vago- and sympatheticotony. Part III, consisting of 143 pages, is take

up with a discussion of special intestinal diseases, and for the most part is much more creditably handled than is Part II. There is no space allotted to a discussion of diseases of the mouth, the esophagus, the pancreas, and the biliary apparatus in their direct or associated influences on gastro-intestinal digestion.

The author shows commendable familiarity with the foreign literature, especially from German sources, but has paid very little attention to the marked advances in and the many contributions to the subject which have recently been developed by American writers. For a book published as late as the current year it is almost unpardonable that no mention should be made of newer methods of diagnosis by fractional gastric analyses, gastric and duodenal sediment studies, and the development of some of the more recent biological and physiological chemical tests, useful in differential diagnosis. The book is presented in a literary style which is somewhat too refreshingly breezy in its use of slang expressions that seem out of place in such an otherwise dignified treatise, and the reviewer occasionally found himself lost in a maze of badly constructed sentences, for instance the following: "This peristaltic action of the antrum plays a very important part in the act of vomiting. At first the muscular tone of the fundus relaxes, leaving its walls flabby; the cardia opens and the waves of the antrum continue, but against a closed pylorus and a relaxed fundus, in which way the contents of the stomach, perhaps aided by the pressure of the diaphragm and the abdominal walls, with mouth open, posterior nares and glottis closed, as in swallowing, are forced out of the mouth."

From the publisher's stand-point the book is attractively bound and printed, and the somewhat mediocre illustrations have been capably reproduced. B. B. V. L.

Skin Cancer. By Henry H. Hazen, A.B., M.D., Professor of Dermatology in the Medical Department of Georgetown University; Professor of Dermatology in the Medical Department of Howard University; Some Time Assistant in Dermatology in the Johns Hopkins University; Member of the American Dermatology Association. Pp. 251; 98 illustrations. St. Louis: C. V. Mosby Company, 1916.

The attractive little book of Hazen's with the title of *Skin Cancer* has been read from cover to cover with great pleasure and interest by the reviewer. The subject under consideration has been divided into nineteen chapters: general considerations; precancerous dermatoses; epithelial warts; basal-celled carcinomata; cubo-celled carcinomata; spino-celled carcinomata; benign and

malignant tumors of the cutaneous appendages; multiple benign tumors; nevocarcinomata and malignant melanomata; carcinoma en cuirasse; endotheliomata; benign connective-tissue tumors; sarcomata and sarcoids; lymphomata; differential diagnosis; prognosis; prophylaxis; treatment; tumors according to location.

The volume is well illustrated and the various histopathological cuts emphasize clearly the various portions of the skin from which the growths may arise. The classification of these various growths has always been an extremely difficult matter and the writer has been extremely clear in his differentiation of the various types. Emphasis has rightly been given to the fact that the type of treatment should depend upon the character of the cells comprising the tumor and the portion of the skin from which they arise. The dictum is again broached that lesions which eventually may give rise to malignancy should be removed in their incipiency. The suggestion might be offered that the various sarcoids are probably of tubercular origin and therefore should be removed from a future edition.

The book is commended to all those interested in the scientific and also practical side of skin cancer. J. C. K.

ANLEITUNG FÜR DIE KRIEGSCHIRURGISCHE TÄTIGKEIT. Von Prof. Dr. LOTHAR DREHER, Leiter der Orthopäd; Abteilung und Oberärzt an der chirurgischen Universitätsklinic in Breslau. S. 158, mit 132 Abbildungen. Berlin: S. Karger, 1916.

THIS war brochure is divided into two parts. After an introduction the general part deals with the subjects of organization, variety of weapons, disinfection in the field, dressings, infection, general effects of gunshot injuries upon the body, narcosis, and local anesthesia. The special part covers gunshot wounds of the extremities, skull, face, neck, thorax, spine, abdomen, and urinary tract, and then concludes with frost-bites, appendicitis, suppurative lymphadenitis, furuncle, and carbuncle. Each subject is considered under two headings—first aid and later treatment.

There is no subject less desirable to commit between the covers of a bound book than that of the surgery of the present war, for revolutionary advances are daily being made, based upon the try-out of different methods by 100,000 surgeons on the various battle fronts, the discovery of new methods by investigators like Carrel and Dakin, and the constant sifting of data by collaborators to establish the best means of procedure. Thus in the book before us the sole treatment recommended for gas phlegmon is the radical and obsolete one of amputation. The prophylactic treatment of tetanus is not referred to, yet since the routine use of tetanus anti-

toxin for a year and a half there have been practically no cases of tetanus treated in France or England. Nitrogen-gas compression of the lung, a valuable procedure in selected cases of continuing hemothorax from lung injuries, is not mentioned. That sovereign remedy for shock—direct blood transfusion—is omitted from the therapeutics of shock, as is its efficient substitute, intravenous saline infusions with adrenalin. Heart massage in emergencies is not described. Trepanation by means of hammer and chisel, as recommended in the book, is a perfectly good way of increasing shock already present. Auscultation is a much gentler method than manipulation in the diagnosis of complete fracture of a rib.

The more one considers the matter the less reason one sees for the publication of these war manuals, from the practical stand-point at least, although they doubtless have their place in a course of study at a military medical school. The surgical teachings from previous wars are being daily controverted, as Fauntleroy has recently shown in reviewing the surgical lessons of the European war. The outstanding feature of all serious wounds in this war is the mutilating and devitalizing effect on the tissues due to large and small irregular pieces of metal, the majority of which are fragments of the high-explosive shell and hand grenade.

As previously pointed out in these columns, in the penetrating and perforating injuries of the abdomen the field surgical experiences of the South African war seemed to prove conclusively that opium, starvation, and rest in the Fowler position yielded better results than treatment by operation. It would seem, from reports, that the contrary is the case in the present war. Where it has been possible for the patient to receive prompt attention the results from operative treatment have been most encouraging in improving the statistics as compared with the expectant line of treatment. As Carrel has rightly stated: "The future of the wounded depends upon the rapidity of transportation and the possibility of treating the wounded as soon as possible."

We hope that we have succeeded in pointing out the uselessness of these manuals in the heat and turmoil of surgical work in the field. That they have a distinct place as collateral reading for the first-aid instructions now being published in the daily press, will not, however, be gainsaid. P. G. S., Jr.

Hay Fever: Its Prevention and Cure. By W. C. Hollopeter, M.D., Attending Physician to St. Joseph's Hospital, etc. Pp. 292; 5 illustrations. New York and London: Funk & Wagnalls Company, 1916.

This treatise on hay fever is a rather conglomerate compilation of antique, medieval, and modern views on the etiology, pathology,

and treatment of hay fever, accompanied by a very complete bibliography covering the literature from 1565 to 1916. The opinions of various authors are quoted freely even to the insertion of extensive abstracts from leading articles. In spite of this elaborate consideration of the literature the author's own conception of the etiology and pathology of hay fever is, to say the least, most confusing. Also, his proposition that local treatment of the nasal mucosa and the nasopharynx with douching and swabbing will effect a cure in the large majority of cases seems a rather extravagant claim. As a book for reference this work will be found most useful, but as a text-book it is too confused and misleading. G. B. W.

THE DESCRIPTION OF AN OPHTHALMOSCOPE. A TRANSLATION OF VON HELMHOLTZ'S "BESCHREIBUNG EINES AUGENSPIEGELE." By THOMAS HALL SHASTID, A.B., A.M., M.D., LL.D., F.A.C.S. Chicago: Cleveland Press.

FEW medical instruments have served their purpose better or fulfilled the early predictions of their originators to a greater extent than the ophthalmoscope. When one pauses to consider the knowledge and facts that have been contributed to medical subjects through its use it is nothing short of remarkable that sixty-five years should elapse before there should appear a translation of von Helmholtz's original description. For his successful efforts in placing this historic and epoch-making contribution at the disposal of many who otherwise would have been deprived of the privilege of reviewing it, Shastid deserves the thanks of the English-speaking profession. The small volume contains but thirty-three pages, the subject being discussed under four headings; (1) illumination; (2) production of a distinct image of the retina; (3) description of the ophthalmoscope; (4) viewing the retina and the image of the flame. The frontispiece shows a reproduction of the original illustration in von Helmholtz's *Beschreibung eines Augenspiegels*. T. B. H.

HARVEY'S VIEWS ON THE USE OF THE CIRCULATION OF THE BLOOD. By JOHN G. CURTIS, M.D., LL.D., formerly Professor of Physiology, Columbia University, in the City of New York. Pp. 189; 4 illustrations. New York: Columbia University Press.

THE author bases his consideration on very thorough translations of Harvey's Latin treatises. Harvey's private lecture notes are also freely quoted, as well as several other Latin and some Greek writings. Harvey's references are traced to their source.

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It will be recalled that in 1602, when Harvey received his doctor's degree at Padua, Galen and Aristotle were still undisputed authorities. It was against the accepted thories, and such other obstacles as weak magnifying glasses, that the "discoverer of the circulation" had to contend. Moreover, much that he could demonstrate he could not explain. Contrary to the other eminent men of his day, Harvey was not dazzled by the past, although he admired it. All these facts are clearly brought out in several chapters.

It is interesting to the present-day physiologist to find some of the theories held by Harvey on the cause of the heart beat. Thus: "I judge the cause of the diastole and expansion to be the innate heat and expansion to occur first in the blood itself, gradually thinned and swelling up like matters in fermentation." The book abounds with such delightful contrasts to modern teaching while showing with what clearness the mind of a great observer grasped the fundamentals of the problem of circulation. A. G. M.

MEDICAL LECTURES AND APHORISMS. By SAMUEL GEE, M.D., Fellow of the Royal College of Physicians; Honorary Physician to H. R. H. the Prince of Wales, and Consulting Physician to St. Bartholomew's Hospital. With Recollections by J. WICKHAM LEGG. Pp. 408. Henry Frowde, Oxford University Press; Hodder & Stoughton, Warwich Square, London, E. C.

THIS book has no new ideas to present. It is, however, very entertaining reading and contains a great many wise sayings. Dr. Gee was of the old school, well versed in the literature of the ancients, eminently observant, of an analytical mind, and at the same time opposed to experimentation as a method of studying nature. He on several occasions refused to give the Harveian oration because it imposed upon the orator the condition of finding out the ways of nature by experiment. Yet he has presented here a work not only of literary charm but of value because of its searching analysis of human nature, its therapeutic suggestions, and above all its presentation of certain medical problems from the purely practical point of view. T. G. M.

WISH FULFILMENT AND SYMBOLISM IN FAIRY TALES. By DR. FRANZ RICKLIN, Zurich. Authorized translation by DR. WILLIAM A. WHITE, of Washington, D. C. Pp. 90. New York: The Nervous and Mental Disease Pub. Co.

THIS is a very interesting Freudian interpretation of fairy tales. According to the author all fairy tales have as their object the

accomplishment of certain wished for results. There is always someone in the tale who will marry either a prince or a queen and according to the Freudian views the structure of these tales is analogous or stands in close relationship to dreams, hysteria or similar symbolic features found in some forms of mental disease. Of course, the author proves to the satisfaction of anyone who believes in Freudian psychology that the leading motive is the sexual. Those who do not believe in Freudian psychology will perhaps deem it a pity that perfectly good fairy tales should be analyzed and laid bare and given a sexual meaning. However, whether one believes in it or not the book is very interesting reading, and once begun the reader is bound to finish the volume.

T. H. W.

INJURIES OF THE EYES, NOSE, THROAT AND EARS. By A. M. RAMSAY, M.D., F.R.F.P.S., Ophthalmic Surgeon, Royal Infirmary, Glasgow; J. D. GRANT, M.D., F.R.C.S., King George Hospital, London; H. L. WHALE, M.D., F.R.C.S., Captain R.A.M.C. (T.F.), the County of London War Hospital, and C. E. WEST, F.R.C.S., Aural Surgeon to St. Bartholomew's Hospital. Pp. 160; 11 illustrations. London: Oxford University Press. Published by the Joint Committee of Henry Frowde and Hodder & Stoughton.

THIS book is a valuable pocket primer not only to the army field surgeon, but to all interested in first-aid work. It is based chiefly upon the experience gained at the front in the base hospitals of the present European war.

There are sixty-five pages devoted to the injuries of the eyes, the chapter on amblyopia due to traumatic neurosis being of special value to the neurologist. The treatment of wounds is presented in a practical manner, the methods mentioned being only those that were found to meet all requirements at the front.

It is to be regretted that more space is not given to injuries of the ear, at least in proportion to that given the other headings. The differential diagnosis between nerve deafness and that caused by an obstructive lesion is rather lightly dealt with. B. D. P.

THE STRETCHER BEARER. By GEORGES M. DEPUTY, M.D. Pp. 138. London: Oxford Medical Press.

THE importance of first-aid stretcher drill has prompted the author to publish this collection of illustrations of the method of handling and carrying the wounded. It serves as an addition to the *Royal Army Medical Corps Training Book*. G. M. L.

INJURIES TO JOINTS. By MAJOR ROBERT JONES, R.A.M.C. (T.). Pp. 189; 29 illustrations. London: Oxford Medical Press.

IN this day of "preparedness," when practitioners are studying some of the chapters of military surgery, this volume by Robert Jones is highly to be commended. The earlier chapters, dealing with the general principles of injuries to joints, their symptoms and treatment are written in a style that is a model for surgical teaching. The reasoning is based on anatomy and pathology and is clear and logical. There follows a systematic review of the various joint injuries and fractures and a definite plan of treatment is given. The book is remarkably free from the vague statements usually found in treatises on joints. G. M. L.

GUNSHOT WOUNDS OF BONES. By CAPTAIN ERNEST W. HEY-GROVES, R.A.M.C. (T.). Pp. 134; 34 illustrations. London: Oxford Medical Press.

MODERN methods of treatment have been under trial sufficiently long for the author to present in text-book style those that have proved most applicable to gunshot injuries in military surgery. Necessity has led to the development of ingenious applications for the immobilization of fractures since the bone plate has failed and the bone graft can rarely be used on account of infection.
G. M. L.

PAINLESS CHILDBIRTH EUTOCIA AND NITROUS OXIDE-OXYGEN ANALGESIA. By CARL HENRY DAVIS, A.B., M.D., Associate in Obstetrics and Gynecology, Rush Medical College, in affiliation with the University of Chicago; Assistant Attending Obstetrician and Gynecologist to the Presbyterian Hospital, Chicago. Pp. 134. Chicago: Forbes & Co., 1916.

THIS interesting monograph is an authoritative statement of the value of nitrous oxide oxygen analgesia in labor. A short history of anesthetic agents and their use in labor is given and the relative merits and dangers of each are discussed. The technic of the administration of this particular form of analgesia is described in detail. The obstetrical results in a group of parturient women so treated are given and contrasted with the results in a group delivered under average normal conditions. It was found that labor was shortened 25 per cent., and that no harm resulted to mother or child from its use. Properly administered, the cost was comparatively slight and a safe and successful analgesia easily secured. The book will be of interest to all concerned with this problem of obstetrics. P. F. W.

PROGRESS
OF
MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF
W. S. THAYER, M.D.,
PROFESSOR OF CLINICAL MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND,
AND
ROGER S. MORRIS, M.D.,
FREDERICK FORCHHEIMER PROFESSOR OF MEDICINE IN THE UNIVERSITY OF CINCINNATI, CINCINNATI, OHIO.

The Epidemiology of Lobar Pneumonia.—Of all acute infectious diseases lobar pneumonia is responsible for the greatest number of deaths in the United States. Previous observations by Dochez and Avery strongly suggest that in most cases the infection is acquired by transmission of the infectious agent from a recovered case which still harbors in the mouth the organisms responsible for the disease. The present study undertaken by STILLMAN (*Jour. Exper. Med.*, 1916, xxiv, 651) was made to determine the following points: first, to study the varieties of pneumococci present in the cases of lobar pneumonia admitted to the Rockefeller Hospital during the past four years; second, a study of the different types of pneumococci present in the mouths of normal individuals; third, to determine how often the disease-producing types of pneumococci occur in the mouths of normal persons; fourth, to determine if possible the frequency of the occurrence of the disease-producing types in the mouth secretions of healthy people associated with cases of pneumonia and, finally, to determine the period of time during which convalescents harbor in their mouths the types of pneumococci which have been responsible for their disease. The results which can be briefly summarized, are in general a confirmation of the previous observations reported from the Rockefeller Institute. The commonest types of organisms responsible for lobar pneumonia are those of groups 1 and 2. The types of pneumococci most frequently found in the mouths of healthy people are those of groups 3 and 4, the latter especially. These organisms are responsible for the minority of cases of lobar pneumonia. Intimate association with cases of lobar pneumonia may lead to the harboring in the mouth secretions of healthy people of the highly parasitic pneumococci or groups 1 and 2. In

addition some evidence was secured to show that true carriers of groups 1 and 2 may be encountered in individuals who have been in no contact with an infected patient, so far as is known. There can be no doubt but what convalescents from lobar pneumonia may carry for long periods of time in their mouths the type of pneumococcus with which they were infected. These facts may throw much new light upon the epidemiology of this disease and subsequently bring to light very important factors concerned in the transmission of pneumonia from one individual to another.

The Influence of Age upon the Venous Blood-pressure in Man.—D. R. HOOKER (*Am. Jour. Physiol.*, 1916, xl, 43) has recorded observations on the venous blood-pressure in normal individuals at different ages. The author used the method he described in 1914 (*Ibid.*, xxxv, 73), the pressure being recorded in centimeters of water. The subjects were grouped in decades, and each figure represents the average of determinations on at least 50 individuals. Cardiac disease was excluded in the subjects studied. "Because of the uncertainties of illumination, the pressures were read at complete collapse of the vein and not, as is more accurate, at the point at which the shadow comes and goes with slight oscillations of the outside pressure." The values are therefore some-what high: 5 to 15 years, 8.30 cm.; 15 to 25 years, 12.66 cm.; 25 to 35 years, 15.00 cm.; 35 to 45 years, 17.98 cm.; 45 to 55 years, 19.04 cm.; 55 to 65 years, 24.17 cm.; 65 to 75 years, 25.59 cm.; 75 to 85 years, 26.00 cm.

The Effects of the Subcutaneous Injection of Organ Extracts upon the Flow of Pancreatic Secretion.—J. ROGERS, J. M. RAHE, G. G. FAWCETT AND G. S. HACKETT, (*Am. Jour. Physiol.*, 1916, xl, 12) report studies made on the dog on the effect of aqueous extracts of organs upon the flow of pancreatic secretion, which they summarize as follows: The effect of the subcutaneous injection in dogs of the residue, or non-coagulable portion, of an aqueous extract of the liver is the immediate and vigorous stimulation of the external secretion of the pancreas. The residues of the thyroid and thymus produce a somewhat less vigorous and later response. The residue of the pituitary and parathyroid glands and of the spleen and pancreas are inert. The residue of the adrenal gland, like adrenalin, vigorously inhibits the intestinal secretions of the pancreas. Only the residue or non-coagulable portion of an aqueous extract of the above mentioned organs shows any appreciable effect upon the intestinal secretion of the pancreas.

The Nature of the Active Principle of the Retroperitoneal Chromaffin Tissue.—M. E. FULK and J. J. R. MACLEOD (*Am. Jour. Physiol.*, 1916, xl, 21) refer to the theory that all chromaffin tissue, whether contained in the suprarenal gland or not, yields adrenin, or a substance having a similar pharmacodynamical action. Vincent has pointed out that this theory is based upon the provisional assumption that chromaffin tissues are specific in their nature and everywhere of the same essential character. In support of this hypothesis, Biedl and Weisel have shown that extracts of the retroperitoneal chromaffin tissues in man have the same effect on arterial pressure as extracts of the suprarenal glands. The authors have employed other pharmacodynamical

tests, namely, the action on the spontaneous contractions of the isolated intestinal muscle and on the tone and contractions of the virgin uterus of the rabbit. An inhibition of the former, along with an augmentation of the latter, was taken as positive evidence of the presence of epinephrin. Acid extracts of the retroperitoneal chromaffin tissue of man, the dog, the cat, the rabbit, the guinea-pig, the white rat, the calf, the sheep and the pig were found to have the same physiological action on intestinal and uterine muscle as the active principle of the medulla of the suprarenal glands.

The Movements of the Mitral Cusps in Relation to the Cardiac Cycle.—A. L. DEAN, JR. (*Am. Jour. Physiol*, 1916, xl, 206) has experimented on the cats heart to determine the exact time relation in the cardiac cycle of the closure of the mitral cusps. A human hair has been attached to one of the mitral cusps, the movements of which have been recorded photographically. He has found that the movements of the mitral cusps vary somewhat with the length of the As–Vs interval. When this averages 0.272 second or more, the cusps move ventricleward slighly a short period after the onset of auricular systole. Toward the end of auricular systole they move auricleward quickly and markedly, but not to a position of complete closure. At the onset of auricular diastole the cusps move quickly ventricleward, the rapidity depending upon the existing intra-auricular pressure. When ventricular tonus is low, and when this obtains, there is a rebound of the cusps from the ventricular walls. The valves remain open until ventricular systole begins. At the onset of ventricular systole the cusps immediately move upward to a condition of complete closure, and remain so until ventricular relaxation begins. During active relaxation of the ventricles the cusps move downward to a lower position than they occupied at the beginning of cystole. From this position they gradually float upward during diastasis. The sequence of movements above described also occurs when the As–Vs interval ranges from 0.147 to 0.272 second, except that time is lacking for a complete opening of the valves before ventricular systole again causes their closure. The valves open slightly during the intersystolic period, the extent increasing with the As–Vs interval. When the As–Vs interval is less than 0.147 second the valves are in the process of closing due to the auricular effect when ventricular systole begins. Hence this cardiac event merely completes the closure already initiated by the auricle. There is in this case only a single closure movement, beginning before ventricular systole—a single movement, due in part to auricular contraction and in part to ventricular contraction. It is commonly taught that the mitral cusps are approximated at the onset of ventricular systole. It has been shown in this work that some degree of approximation obtains when ventricular systole begins if the As–Vs interval falls within limits that may be considered normal (0.13 to 0.18 second). It has been further shown, however, that the extent of this approximation is directly related to the length of the As–Vs interval, so that when the As–Vs interval equals 0.272 the cusps are as widely separated as at the onset of auricular systole. It is of practical importance to recognize that in cases of delayed A–V conduction the valves may undergo two distinct movements of closure, the first near the end of auricular systole, the second at the beginning of ventricular systole.

SURGERY

UNDER THE CHARGE OF

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A Study of the Anatomy, Pathology, and Treatment of Uterine Prolapse, Rectocele and Cystocele.—FRANK (*Surg., Gynec. and Obst.*, 1917, xxiv, 42) says that with very few exceptions, anterior and posterior colporrhaphy combined with either the Alexander operation or ventrofixaton are applicable to all cases of prolapse or cysto-rectocele and retroverson both during and after the childbearing period. The technic of these plastic operations can be learned as precisely as that of inguinal hernia, if the student is taught the regional anatomy. To obtain the proper dimensions of the reconstructed canal, however, requires considerable experience and proficiency in operating. The disadvantage of the technic, which is described at length, is the considerable time required. A rapid operator cannot complete currettage, amputation of the cervix, anterior and posterior colporrhaphy, ligation of the tubes, and ventrofixation in much less than ninety minutes. In some cases (though very rarely) it may prove advisable to perform the vaginal plastic and the abdominal fixation at separated sessions (two weeks' interval).

The Actual Cautery in the Treatment of Chronic Ulcer of the Stomach.—SCUDDER and HARVEY (*Surg., Gynec. and Obst.*, 1916, xxiii, 718) investigated experimentally, on dogs, Balfour's method of treating certain chronic ulcers of the lesser curvature of the stomach by the application of the actual cautery followed by suture of the cauterized area. It appears that the suture of the cauterized margins of the stomach wall is attended by practically a normal reparative process similar to the reparative process following a simple incision with the knife. The method is applicable to a chronic ulcer seated upon the lesser curvature so far away from the pylorus as to make its removal by excision difficult. Such an ulcer may be cauterized from the center out, as suggested by Balfour, so that the loss of substance occasioned by the cauterization may be as large as 1½ inches or more in diameter and the edges may be then approximated with the assurance of a proper healing of the wound. Ulcers seated on the posterior wall of the stomach which are safely approached by a gastrotomy incision may have their edges thoroughly cauterized and also the base thoroughly cauterized even when it is adherent to the pancreas, and be sutured with the assurance to the surgeon that the reparative process will proceed satisfactorily. Certain chronic ulcers adherent to the posterior parietics and pancreas do not lend themselves to easy and safe excision and suture. The cautery is sometimes applicable to this special group of cases. It will destroy any beginning cancer in the edges of the ulcer if the cauterization is thoroughly done. In order to close the stomach following a simple excision of a portion of

the stomach wall, a very large wound remains to be sutured. Following the use of the cautery no such large gaping wound exists, and the stomach is closed more readily than after a wide excision by the knife. With cauterization as with simple excision, the stomach should be carefully mobilized and the part to be operated on controlled, so that the portion actually cauterized is rendered accessible. The suture material employed in human cases in each instance has been No. 1 chromic catgut. It has not been found necessary to reinforce the sutured area by interrupted linen suture of the peritoneal surface in all cases. If it is possible to place these interrupted linen sutures it is wise to do so. Following any extensive plastic of the stomach, a gastro-enterostomy should be done (Mayo).

Hemostasis by Interposition of Muscle, Fat and Fascia in Parenchymatous Organs.—RISLEY (*Surg., Gynec. and Obst.*, 1917, xxiv, 85) carried out a series of 12 experiments on dogs to determine the value of the above tissues in stopping hemorrhage in such organs as the kidney and liver, when other forms of hemostasis are either impossible or undesirable. He found that the ideal hemostatic in wounds of parenchymatous organs is interposed muscle taken at the time of operation from the patient's own body. Such muscle in order to most effectively stimulate fibrin formation should be jaggedly cut with a knife and not crushed as with a scissors cut, nor should its hemostatic properties be extracted by its contact with salt solution. Fascia and fat act to a more limited degree as hemostatics; fascia more than fat, but both very much less than muscle. In the liver, however, both fascia and fat seem at times to be very efficient hemostatics. These tissues readily unite to the bleeding surface to which they are sewed, and form a smooth, solid scar. Microscopical examination of specimens removed at varying intervals after operation show, in the absence of sepsis, a beginning transformation of muscle into fibrous tissue, a partial absorption of fat and change into fibrous tissue, no changes in fascial transplants, in practically every case a firm blending of the interposed tissue with the cut surface of the parenchymatous tissue, the formation of new bood channells and no degenerative changes of any note. He concludes therefore, that muscle, fascia and fat can be safely interposed into these tissues and after acting as immediate hemostatics later undergo fibrous change and form a firm union with the parenchymatous tissue.

THERAPEUTICS

UNDER THE CHARGE OF

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Some Studies of Theocin.—CHRISTIAN (*Arch. Int. Med.*, 1916, xviii, 606) says that a fairly complete study of a small group of patients with acute nephritis or chronic nephritis or cardiorenal disease indicates

that theocin in patients with slight or no edema has little or no therapeutic value, inasmuch as diuresis is not constantly produced, elimination of nitrogenous substances quite often is slightly if at all increased and renal function is frequently decreased after giving theocin. In cardiorenal cases with marked edema theocin is of therapeutic value, because it produces, especially in conjunction with digitalis, an active diuresis with increased sodium chloride elimination, which decreases edema, a troublesome feature in these cases. Inasmuch as there is evidence that following an active diuresis renal function is depressed, an intermittent usage of theocin seems preferable to a continuous usage in cardiorenal cases with edema.

The Relation of Diet to Beriberi and the Present State of Our Knowledge of the Vitamins.—VEDDER (*Jour. Am. Med. Assn.*, 1916, lxvii, 1494) says that as there are many conditions under which it is difficult for certain people and institutions to procure a rich and varied diet it is better to give simple dietary rules for the prevention of deficiency diseases. The rules are as follows: In any institution where bread is the staple article of diet, it should be made from whole wheat flour. When rice is used in any quantity, the brown under-milled, or so-called hygienic rice, should be furnished. Beans, peas, or other legumes, known to prevent beriberi, should be served at least once a week. Canned peas or beans should not be used. Some fresh vegetable or fruit should be issued at least once a week and preferably at least twice a week. Barley, a known preventive of beriberi, should be used in all soups. If cornmeal is the staple of diet, it should be yellow meal or water-ground meal, that is, made from the whole grain. White potatoes and fresh meat, known preventives of beriberi and scurvy, should be served at least once a week, and preferably once daily. The too exclusive use of canned goods must be carefully avoided. Vedder is sure that the strict application of these rules will eradicate scurvy and beriberi, and he believes that they would be equally efficacious in eradicating pellagra from the United States.

Studies in Prophylactic Immunization with Bacillus Typhi-exanthematici.—PLOTZ, OLITSSKY and BAEHR (*Jour. Am. Med. Assn.*, 1916, lxvii, 1597) write concerning their experience with prophylactic immunization against typhus fever. The vaccine consisted of a suspension of fifteen strains of B. typhi-exanthematici in physiologic sodium chlorid solution which had been subjected to a temperature of from 58° C. to 60° C., for from half an hour to one hour. After being tested aërobically and anaërobically as to its sterility, the vaccine was then diluted so that each cubic centimeter contained about two billion bacteria, and 0.5 per cent. phenol (carbolic acid) or tricresol added. Three injections consisting of 0.5, 1, and 1 c.c., respectively, were given in five- or six-day intervals. In all, 8420 persons, members of 109 hospital, sanitation and other units in Serbia, Bulgaria, and Volhynia, were vaccinated against typhus fever during the epidemic of 1915–1916, an attempt being made to include in this number only the persons who were most exposed to the danger of infection. Of this number six developed the disease during the four months of the epidemic. Their experiences in the Balkans and Volhynia during the winter and spring of 1915–

1916 with the vaccine made of B. typhi-exanthematici would seem, therefore, to indicate that it is capable of reducing the incidence of the disease, although it does not produce an absolute immunity to typhus fever.

The Comparative Resistance of Bacteria and Human Tissues to Certain Germicidal Substances.—LAMBERT (*Jour. Am. Med. Assn.*, 1916, lxvii, 1300) says that it is recognized that an ideal germicide for use on infected tissues of the body is one that will kill the pathogenic microörganisms present without at the same time injuring the tissues. Lambert found that cells are more easily destroyed than bacteria by all but one (iodin) of the agents used. However, in the case of several, mercuric chlorid, sodium hypochlorite (Dakin's solution), potassomercuric iodid, argyrol and phenol—the difference is not great. It is interesting that no harmful effect whatever was seen from exposure of cells for one hour to 10 per cent. alcohol. The low bactericidal power of hydrogen peroxid, so popular among the laity, is noteworthy. It is possible, however, that on account of the fibrin-dissolving property of iodin, causing conceivably an inhibition of wound healing, some other substance may be found approaching more closely the ideal tissue disinfectant.

Alpha-iodin, Active Constituent in Thyroid.—KENDALL (*Boston Med. and Surg. Jour.*, 1916, clxxv, 557) reports his investigations which seem to indicate that the active constituent of the thyroid gland is contained in alpha-iodin, a crystalline compound. There appears to be no other substance in the thyroid secretion which acts directly. This substance, given even in very small amounts, will supplant thyroid activity, relieving the conditions of myxedema and cretinism, and in excess will produce symptoms simulating exophthalmic goitre. It appears to have no direct action on the pulse rate. The extent to which the rate is affected depends not on the administration of the thyroid, but on the simultaneous ingestion of food, and in particular of amino-acids. This effect may be outlined as follows: After the administration of the compound, there is no apparent effect for many hours. There is no increased pulse rate, nor drop in blood pressure. However, if the thyroid hormone and amino-acids are injected simultaneously, the pulse rate is enormously affected, and even death may result, due to the apparently great increase in metabolism going on in the animal. It appears very probable that the thyroid hormone manifests its activity by reacting in some way with amino-acids.

The Toxicity of the Present Supply of Salvarsan and Neosalvarsan.—ORMSBY, MITCHELL, MOODY and ELLIS (*Jour. Am. Med. Assn.*, 1916, lxvii, 1756) all report a striking increase in the number of severe reactions following the injection of salvarsan and neosalvarsan recently imported from Germany. The toxic symptoms following salvarsan were strikingly vasomotor and accompanied or followed the injections. Marked generalized erythema, and in some cases urticarial wheals, appeared. Lacrimation occurred, followed by injection of the conjunctival vessels. The patient tossed the head from side to side in evident

respiratory embarrassment, and complained of a feeling of constriction about the chest. The pulse, at first full, quickly became rapid and weak, accompanied usually by pallor. In a few cases, nausea and vomiting occurred. The reaction following the injection of the present neosalvarsan differs from that of salvarsan in that there is a marked tendency to nausea and vomiting. In a few cases urticarial wheals have followed each injection, but the diffuse erythema, lacrimation and respiratory difficulty with fall of blood-pressure, observed after salvarsan, have not occurred.

The Administration of Arsenobenzol by Mouth. — SCHAMBERG, KOLMER and RAIZISS (*Jour. Am. Med. Assn.*, 1916, lxvii, 1919) say that experiments on animals have demonstrated that arsenobenzol (salvarsan) can be administered by mouth in solution as in capsules and become absorbed into the blood. The proof that absorption takes place is evidenced by the fact that a distinct destructive influence on trypanosomes in the blood of experimentally infected animals is exerted. Experimental studies on animals also demonstrated that arsenobenzol can be administered in capsule form over long periods of time without harmful results. Clinically they report that, during the course of four months, arsenobenzol in capsule form has beem administered by them to about thirty patients in various stages of syphilis. The drug was given at first in ordinary gelatin capsules, but later in luteric capsules, in order to prevent the capsules being absorbed in the stomach. In a general way they found that the effect of the drug on the secondary eruption is much slower than after intravenous injection. Ordinarily it required about three weeks to cause the disappearance of a macular or papular eruption with the doses administered. In 1 case, however, a large papular syphilid of the face disappeared in three or four days, with almost the degree of rapidity that it would after an infusion of the drug into the blood. Tertiary nodular lesions required from two to three weeks' administration to affect their disappearance. In order to prevent oxidation of the arsenobenzol in the capsule, sodium hydrosulphite was incorporated with it, and a small amount of bismuth subgallate was added to prevent diarrhea. A capsule containing one-half grain of arsenobenzol was given after each meal, and in most patients no gastric or intestinal distress resulted. Some patients were able to take the capsules for six weeks without any distress. The authors do not advocate the use of salvarsan by mouth as a substitute for intravenous or intramuscular administration. It is not always possible, however, to administer salvarsan by intravenous or intramuscular injection. Inaccessibility of veins by excessive adiposity, thrombosis of veins due to repeated injections, or similar reasons may prevent intravenous injections. Another indication for the use of salvarsan capsules is as a supplementary treatment to intravenous administration. The disadvantage of intravenous salvarsan therapy is the fact that most of the drug is eliminated in a short time, but by combining the oral administration of salvarsan with the intravenous, a continuous source of the drug may be supplied to the blood. If it becomes necessary to administer a drug by mouth for the treatment of syphilis—the authors advocate salvarsan in preference to mercury, first, because it is a more powerful spirocheticide, and second, because it has a more

tonic influence than mercury. The cost of the drug might readily be a deterrent factor in prescribing salvarsan by mouth, as much larger doses are required to produce the same effect, in comparison with more efficient routes of administration. This method of treatment is to be reserved for special conditions and for particular cases.

OBSTETRICS

UNDER THE CHARGE OF

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Pregnancy Complicated by Parovarian Cyst. — AHLSTRÖM (*Jour. Svenska Läkaresallskapets Handlingar, Stockholm*, No. 3, 1916) reports the case of a pregnant woman at seven months who had a parovarian tumor which became twisted and incarcerated. The tumor had been pushed into Douglas's pouch by the pregnant uterus, and it is thought that the passage of hard fecal lumps through the rectum had resulted in twisting the tumor on its pedicle.

The Part Played by the Thymus Gland in Sudden Death in Newborn Infants.—HAMMAR (*Jour. Svenska Läkaresallskapets Handlingar, Stockholm*, No. 3, 1916) publishes the results of his study of microscopic sections of the thymus taken from sixteen children, some of whom are in the first days of life. Hammar, two years ago, published the results of his examination of the thymus in 14 cases of sudden death from various causes. The results of his studies lead him to believe that sudden death in the newborn does not result from active swelling of the thymus or any other condition of this gland. The thymus is often large in the newborn, but there is no evidence that this influences the vital state of the infant.

Malignant Chorio-epithelioma. — ENGSTRÖM (*Finska Läkaresallskapets Handlingar*, No. 8, 1916) previously reported 5 cases of malignant chorio-epithelioma of the uterus. He adds to this the results of minute study in a sixth case. In this patient the uterus and adnexa and part of the pelvic cellular tissue were removed. Metastases followed in the pelvis, liver, and left lung, from which the patient died. In this case four years had passed since the previous pregnancy which was the patient's fifth. The question naturally arises whether it is possible for fetal cells to remain latent in the uterus for four years before developing. Cases reported in the literature would indicate that this is possible. In one an interval of nine years had elapsed between pregnancies. So far as the best reported cases are concerned, it cannot be proved that this condition can remain latent so long, and the most natural explanation is found in the fact that a patient may be pregnant without suspecting it, the pregnancy terminating in a very early abortion.

Sterilization by Cautery Stricture at the Intra-uterine Tubal Openings.—Dickinson (*Surg., Gynec. and Obst.*; August, 1916) describes a method which he had devised and employed for sterilization which avoids direct surgical interference. It must first be ascertained that the patient is free from any condition which requires hysterectomy. The time chosen is from seven to ten days after a period. With the patient upon the back and the Sims speculum in place, the anterior lip of the cervix is seized by tenaculum forceps and held steady. From 5 to 10 minims of a 10 per cent. solution of novocain in adrenalin solution are then injected into the uterus and held there under pressure for a few seconds. Ten minutes are allowed for the anesthetic effect. The uterine canal is then wiped with Churchill's tincture or pure carbolic acid. The ordinary uterine sound, thoroughly sterilized, is then introduced and passed to the fundus and turned sidewise to find the cornu, and the exact distance of the cornu is noted. The cautery sound is then bent to fit the uterine sound, and has its slide pushed up so that the portion of the sound remaining exposed exactly equals the measurements of the uterine cavity already made by the sound. The cautery sound has a platinum point which can be heated by an electric current. When this is turned on the sound is applied against the cervix in plain sight, and the time is noted required to burn into the tissues enough to bury the platinum wire. The current is then turned off, the sound allowed to cool, and passed into the uterus to the cornu, then the current is turned on and the tissues at the cornu are cauterized by the heated platinum wire. Very little discomfort usually follows this application and the same procedure is carried out on the opposite cornu. On the cervix a slough forms which separates, leaving a clean granulating wound and this serves as an index of what is going on within. About three or four months after this procedure the patient may be examined by the roentgen-ray and a positive diagnosis made of closure of the tube by scar tissue. The writer describes similar procedures in other portions of the body where cauterization has been useful, and reasons by analogy that it will bring about the desired result where sterility is the object in view.

Hemolytic Anemia in Pregnancy.—In the *Ztschr. f. Geburtsh. und Gynäk.*, 1916, lxxi, No. 1, Esch contributes an extensive paper upon this subject. In all he reports 6 cases, 3 of whom died. In three of the patients there was hemorrhage in the retina, and in three the spleen was enlarged. This disease differs essentially from true pernicious anemia although when the blood is examined the results resemble those obtained in pernicious anemia. This develops in the second half of pregnancy, and is essentially a destructive process in the blood. Probably the resisting power normally present in the red corpuscles is greatly reduced or there is a great exaggeration in the natural physiological processes of hemolysis. Owing to the anemia these patients lack oxygen greatly and are partly anesthetized with carbon dioxid as a result. When in labor these patients seem to suffer much less than others. When they begin to improve their progress is rapid and there is usually no return of the anemia. In most cases pregnancy terminates prematurely in spontaneous labor and these patients do not improve until after delivery. The prompt termination of labor is usually the

best treatment and most of the children are lost as they have little power of resistance. Arsenic is the best remedy for this form of anemia. In 5 cases intramuscular injections of blood were given. Two patients seemed much improved after several injections; one had eight in all, 440 c.c. of defibrinated blood, the hemoglobin increase from 9 to 22 per cent. with corresponding improvement in other ways.

Pressure on the Brain as a Cause of Eclampsia.—ZANGEMEISTER (*Ztschr. f. Geburtsh. und Gynäk.*, 1916, lxxi, No. 1) believes that the clinical picture in cases of eclampsia points conclusively to pressure on the brain as the principle cause of convulsions; that treatment most successful in preventing and controlling eclampsia reduces the edema and irritation of the brain. When the uterus contracts the blood-pressure rises and adds to the pressure on the brain, and convulsions occur; on the contrary the blood directly relieves the condition.

Is the Operation of Cesarean Section Indicated in Breech Presentation?—MCPHERSON (*Am. Jour. Obst.*, November, 1916) has examined the records of 3412 cases of breech presentation and delivery among 97,000 confinements. An effort was made to ascertain accurately the cause of fetal mortality in these cases and to exclude all conditions which were not the abnormal presentation alone. As to the frequency of breech presentation, it is approximately 3 per cent. The mortality for the mother is practically that of uncomplicated spontaneous delivery when the head presents. In the series of cases studied the maternal mortality was 0.96 per cent. and was produced by placenta previa, chronic nephritis, chronic endocarditis, pneumonia, and other serious conditions. If these be excluded the mortality from the abnormal presentation only was 0.47 per cent. When the interests of the child are considered the death rate is about 10 per cent., and the difference in mortality between the children of primiparæ and multiparæ was practical nothing. McPherson criticises a recent paper whose author urges Cesarean section for breach presentation in primiparæ. When the cases reported were analyzed one had a submucous fibroid which would have prevented delivery through the vagina had the head presented. In the other case the pelvis was slightly contracted and it was thought that operation was safest for mother and child. While Cesarean section is not indicated for breech presentation only, it may be the safest method of delivery in some breech cases. In primiparæ at beyond the average age of childbirth and in whom the normal mechanism of labor does not develop the risk to the child in delivery in breech presentation is considerable, while the mother must sustain severe lacerations. If the child be large and well developed, in the interests of mother and child section is the safest procedure. So in justominor and rachitic pelves where breech presentation is present delivery by section is safest for mother and child.

Pregnancy Complicated by Pelvic Infection and Septicemia.—MOORE (*Am. Jour. Obst.*, November, 1916) reports the case of a rachitic negress who had a contracted pelvis and was delivered by Cesarean section. Three weeks after operation she developed multiple neuritis from which she finally recovered. In her second pregnancy she came

into the hospital and was delivered of a premature fetus. Two hours after delivery she had a severe chill followed by high fever and this was repeated on the following day. An examination of the blood showed a pure growth of Staphylococcus aureus. The patient made finally a complete recovery.

GYNECOLOGY

UNDER THE CHARGE OF

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Polycystic Kidney.—The results of the clinical experience with this condition at the Mayo Clinic are given in a recent paper by BRAASCH (*Surg., Gynec. and Obst.*, 1916, xxiii, 697). The series comprises 41 patients in whom at operation polycystic kidneys were found, although in only 26 of these had the condition been previously diagnosed. The average age of the patients was forty-six years, but 10 of them were under forty years and 7 were over sixty years. No material difference was noted with regard to sex. The predominant symptoms were pain, hematuria, tumor, and diminished renal function. The pain is not severe, as a rule, unless interference with urinary drainage or wide-spread infection occurs. There may be occasionally a dull pain in either loin, probably due to tension of the cysts. Hematuria occurred in 40 per cent. of the cases. It is evidently caused by the rupture of small bloodvessels in the cysts, and becomes manifest only when one of the cysts ruptures into the renal pelvis. It is usually stopped by puncture of the blood-distended cysts. Tumor was noted in 31 of the 41 patients, but was not always recognized as kidney, being occasionally mistaken for the liver or the gall-bladder. In only about half the cases was a bilateral tumor noted, in spite of the usual bilateral occurrence of the condition. The kidney function was directly estimated by the phthalein test in only 11 cases, being low in nearly all, and showing merely a trace of elimination in 2, both of whom died after operation. A normal phthalein and a normal specific gravity of the urine may be present, however, with polycystic disease, and such findings by no means exclude this condition. The chief value of the phthaleln test is in differential estimation of the two kidneys, a point of great importance in determining upon the advisability of operation. *Treatment:* In 14 cases nephrectomy was done, a surprisingly high number in view of the fact that the condition is nearly always bilateral. The indications were large tumor with complete degeneration of one kidney (4 cases), unilateral lithiasis (3), diffuse infection (3), hematuria (2), traumatic rupture (1), and hydronephrosis (1). There was 1 postoperative and 1 subsequent death; 10 of the remaining patients were traced and found well after periods of one to ten years. The Rovsing operation (multiple puncture of the cysts) was done in 10 patients, with 2 postoperative and 1 subsequent death, the remaining

7 patients being alive at varying periods up to five years. The other case had merely exploratory operations, or the kidney condition was found incident to some other operation. The conclusions drawn by Braasch are briefly as follows: When there is marked clinical evidence of toxemia; when the blood-pressure is increased to 200 or more; when the various functional tests show marked renal disturbance, any operation is attended with considerable danger, and is of questionable value. When, however, there is evidence of but a moderate degree of renal insufficiency the Rovsing operation is followed by considerable benefit. Nephrectomy is indicated only in the presence of widespread infection, persistent hematuria, or destruction of renal tissue from mechanical obstruction, lithiasis, or other complication. It is, of course, possible only after the function of the remaining kidney has been demonstrated to be satisfactory, but under these conditions may be of great benefit.

Radium Treatment of Cancer of the Cervix.—A very brief report upon 9 cases of cervical carcinoma treated with radium at the Clarkson Hospital, Omaha, has recently been published by FINDLEY (*Am. Jour. Surg.*, 1916, xxx, 337). In 5 of these cases a radical hysterectomy was performed, followed, and in some instances preceded also, by the use of radium; in the remaining 4 radium was used without operation. In 3 cases, which were considered inoperable, the condition became frankly operable after the application of the curet, cautery, and radium. In 1 case scrapings from the cervix were of a highly malignant type, but careful search in the removed uterus showed only a nest or two of degenerated epithelium. One case was not favorably influenced by the radium, a recurrence in the vaginal vault manifesting itself shortly after three applications. In 2 cases pain was promptly relieved, in 4 it was only partly or not at all relieved, and in 1 it was apparently intensified by the radium treatment. One patient in whom the radium was used postoperative, developed a vesicovaginal fistula, which, however, promptly healed spontaneously. In one the general peritoneal cavity was invaded by cancer with such rapidity following hysterectomy and radium application as to suggest that the radium might have had a stimulating effect upon the cancerous growth. In 2 cases the patients failed to react well to the radium treatments developing a toxic condition, and it seemed possible that death was hastened by the action of the radium. Only two of the nine patients are still living, after periods of ten and fourteen months. In one there is no evidence as yet of recurrence, while in the other enlarged lymph nodes have lately developed in the right iliac region, and are strongly suggestive of recurrence. While the record here outlined certainly cannot be considered a very brilliant one, Findley thinks it fair to conclude that radium has a place in the treatment of cancer of the cervix, since it probably prolongs life in nearly all instances, and converts many inoperable cases into operable ones. The radium was applied in the cases discussed above in doses of from 32 to 62 mg., in properly screened tubes in the vagina, this being often supplemented by 20 mg. on flat applicators placed on the abdomen for the purpose of cross fire.

OTOLOGY

UNDER THE CHARGE OF

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EMERITUS PROFESSOR OF OTOLOGY IN THE HARVARD MEDICAL SCHOOL, BOSTON.

A Case of Herpes Zoster Oticus.—By CAPTAIN ARCHER RYLAND (*Jour. Laryngol., Rhinol., and Otol.*, vol. xxxi, No. 12).—The patient, a man, was admitted to the Cambridge Hospital, at Aldershot, with the following symptoms: Pain in the depth of the left ear, aching pain and swelling of the left ear itself, slight impairment of hearing, but no discharge from the middle ear, weakness of the left side of the face, sensation of soreness on the left side of the throat and pain on swallowing, lachrymation of the left eye, dizziness with perception of rotation of objects from left to right. These symptoms, which followed each other in rapid succession, appeared in the following order: pain on the left side of the throat, pain and throbbing in the depth of the ear, burning pain, edematous swelling and livid discoloration of the auricle, followed by the appearance of herpetic vesicles on the floor and posterior wall of the left cartilaginous meatus, subsequent appearance of similar vesicles over the upper portion of the sternomastoid muscle and, later, left facial paralysis with vertigo and nausea. The facial paralysis occurred twenty-four hours after the vesicular eruption; throughout the illness the pulse was slow and the temperature subnormal, and dizziness and nausea were pronounced symptoms. On examination the left drum-head was found to be intact. There was some injection of the manubrial plexus and, in a lesser degree, of adjacent portions of the membranæ vibrans. There was no sign of increased tympanic pressure and there was an impaired audition for the lower fork tones and for the upper tone limit on the tone scale. The whole of the auricle was red and swollen with diminished cutaneous sensibility, and that part of the herpetic eruption which included the external ear was confined to the floor and posterior wall of the soft external meatus and of the conchæ. Discreet herpetic vesicles were present in addition on the skin overlying the mastoid process, with a similar slightly marked eruption distributed over the upper fourth of the sternomastoid and a few vesicles on the anterior surface of the left posterior faucial pillar. The anterior third of the left half of the tongue showed a few non-vesicular red spots, and there was some tenderness over the included area. These symptoms indicated implication of the following cranial nerves: Seventh, left facial paralysis not quite complete as regarded the orbicularis palpebrarum. Eighth, impaired audition but, at the time of admission, no signs referable to the vestibular ganglion. Ninth, disturbance of common sensation of the anterior third of the left half of the tongue, but no disturbance of taste. There was no spontaneous nystagmus, abnormality of pupils or of ocular movements of affection of the larynx. Six days after admission the auricle had recovered and its cutaneous sensibility had become normal,

the herpetic eruption was rapidly clearing, but remained most persistent on the meatus, the spontaneous nystagmus was still faintly present, the vomiting was less frequent, there was still some dizziness, the facial paralysis remained unchanged, and the leukocytes count was 8000 and the spinal fluid healthy. One month from the onset of the attack the facial paralysis was slowly recovering, there was a moist eczematous area corresponding to the region of the herpetic eruption, and some remaining impairment of hearing of the middle-ear type. The emphatic points in this case are, its suggestion in the first few days of the disease of an acute intracranial complication and the determination of the diagnosis by the early appearance of the herpetic eruption, which lasted for eight days following an auriculotemporal distribution, during the major portion of which from the beginning there was, in addition to the nausea, intense, frequent and uncontrollable vomiting, suggesting ganglionic involvement of the vagus; the spontaneous nystagmus made its appearance on the third day and its total duration was twelve days.

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

MILTON J. ROSENAU, M.D.,

PROFESSOR OF PREVENTIVE MEDICINE AND HYGIENE, HARVARD MEDICAL SCHOOL, BOSTON, MASSACHUSETTS,

AND

JOHN F. ANDERSON, M.D.,

FORMERLY DIRECTOR OF THE HYGIENIC LABORATORY, PUBLIC HEALTH SERVICE, WASHINGTON, D. C.

The Etiology of Acute Epidemic Poliomyelitis.—MATHERS (*Jour. Infec. Dis.*, 1917, xx, 114) examined bacteriologically fresh material from 10 cases of acute poliomyelitis and isolated a peculiar polymorphic streptococcus-like organism in nine instances, in seven of which the growth has been pure. Similar organisms have been demonstrated microscopically in the tissues of the central nervous system of these cases. Cultures of this coccus injected into rabbits have produced paralysis of various groups of muscles, and characteristic lesions in the central nervous system consisting of hyperemia and edema of the tissues, with hemorrhages, round-cell perivascular infiltration, and neurophagocytosis in the spinal cord, especially in the gray substance, similar in every detail to the changes considered characteristic of acute poliomyelitis in man. This micrococcus has been recovered from the lesions in the inoculated rabbits by both cultural and microscopic methods. The artificial cultivation of the poliomyelitis coccus in an ascites-fluid tissue medium under anaërobic conditions causes changes in the media which cannot be differentiated from those previously described for cultures of the so-called virus of poliomyelitis. Morphologically, also, this bacterium when grown on the same media is

similar to the virus, and in stained smears it appears in minute Gram-positive coccus-like bodies arranged in pairs, groups and chains. These minute forms disappear when the organism is cultivated in other media under aërobic conditions. The morphologic, cultural, and pathogenic characters of the poliomyelitis coccus thus far determined indicate that it is an important factor in the disease.

Studies on Immunity in Typhus Exanthematicus with Reference to the Antibodies in Man and Guinea-Pig Demonstrable by the Dale Method.—DONZER and OLITSKY (*Jour. Infec. Dis.*, 1917, xx, 99) state that antibodies against Bacillus typhi-exanthematici, demonstrated by the Dale method, are found in the serum of typhus-fever patients after the crisis. These antibodies are not present in the serum during the height of the disease. Antibodies against Bacillus typhi-exanthematici are found in the cells of typhus-fever guinea-pigs after the crisis. These antibodies are not demonstrable during the height of the disease. The reaction in the serum of typhus-fever patients and in the cells of the guinea-pig is specific; that is, by the use of similar methods no antibodies to Bacillus typhi-exanthematici could be demonstrated in the blood of normal individuals or of patients suffering from other infections. Guinea-pigs in the postcritical stage of typhus fever showed antibodies only to Bacillus typhi-exanthematici, and not to Micrococcus anureus, Bacillus typhosus, or Bacillus acne. The results with the Dale method offer further evidence of the etiologic relationship between Bacillus typhi-exanthematici and typhus exanthematicus.

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Digestibility of Very Young Veal.—LANGWORTHY and HOLMES (*Jour. Agricul. Research*, 1916, vi, No. 16) state that throughout the United States little was known until recently regarding very young veal, since the sale of calves less than three to six weeks old for food is prohibited by Federal and State laws. Our attitude toward veal, as toward many other foods, has been determined in part by custom and prejudice

and in part by economic conditions and experience, often being illogical; therefore it is of interest to ascertain in such cases how far belief is justified by facts as shown by controlled experimental tests. That the common opinion that veal is less wholesome than beef and young veal less so than mature veal is not a consistent prejudice against young flesh foods is shown by the common and apparently growing taste which prefers squab to pigeon, ranks broilers as superior to fowls, considers sucking pig a great delicacy, and regards hot-house lamb—that is, lamb less than three months old and rapidly grown and fattened—as much superior to older lamb as lamb is to mutton. As determined by the experiments herein reported, the digestibility of the protein of bob veal is the same as that found for market veal—namely, 93 per cent., in round numbers. The subjects of both dietary and digestion experiments, so far as could be learned, experienced no physiological disturbances during the experimental period or afterward. The tests showed that such veal can be prepared for the table in palatable ways, and that so far as could be judged it was not unwholesome when eaten in quantity.

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

JOHN McCRAE, M.D., M.R.C.P.,

LECTURER ON PATHOLOGY AND CLINICAL MEDICINE, MC GILL UNIVERSITY, MONTREAL; SOME TIME PROFESSOR OF PATHOLOGY IN THE UNIVERSITY OF VERMONT, BURLINGTON, VERMONT; SENIOR ASSISTANT PHYSICIAN, ROYAL VICTORIA HOSPITAL, MONTREAL,

AND

OSKAR KLOTZ, M.D., C.M.,

PROFESSOR OF PATHOLOGY AND BACTERIOLOGY, UNIVERSITY OF PITTSBURGH, PITTSBURGH, PA.

Lesions in Rabbits Produced by Streptococci from Chronic Alveolar Abscesses.—Noting that there is in the literature no data concerning the result of animal injections with strains of Streptococcus viridans from alveolar abscesses of patients otherwise in perfect health, MOODY undertook such a research (*Jour. Infect. Dis.*, 1916, xix, 415). At the outset, the author states that streptococci from all infectious foci should be studied in this manner, in working toward the proof or disproof of the theories of specificity and elective affinity. Strains of Streptococcus viridans were isolated in pure culture from patients with alveolar abscesses. Out of a total of 55 patients, 49 yielded cultures of Streptococcus viridans free from contamination and suitable for injection. The type of organism, other than that it produced green on blood agar, was never determined. No further cultural reactions were undertaken, so that it is impossible to say exactly what organisms were injected. The patients from whom material was obtained were divided into three groups: Group A, those having chronic alveolar abscesses and

"articular rheumatism;" Group B, those having chronic alveolar abscesses and some systemic disorder other than articular rheumatism; Group C, patients having chronic alveolar abscesses, without evidence of any other illness. Four young rabbits averaging 1000 grams in weight were injected with cultures from each case; two received 1.5 to 2 c.c. of a suspension of the sediment from an eighteen- to twenty-hour growth of the original pus in ascites dextrose broth, and two received a similar injection, the latter culture having been obtained by picking typical green colonies from an original blood agar plate to ascites dextrose broth. In all, 178 rabbits were injected in this manner. The rabbits were killed with chloroform after intervals of five to eight days following injection and autopsied immediately and completely. Full notes were taken during the autopsy. The value of these experiments is enhanced by the well maintained constant factors of cultivation —time of the organism, size of dose, and length of life allowed before autopsy. Lesions were found at autopsy in the stomach, muscles, joints, endocardium, kidneys, and jaws. The frequency was in the order named. It was noted that there was a higher percentage of lesions produced in usual areas of localization by strains from cases showing articular rheumatism than from the other groups. Strains from Group B and C frequently produced hemorrhage of the stomach. This very rarely amounted to true ulceration. Hemorrhage, oftenest near the tendinous end, was seen in the muscles. In the joints, suppuration was seen in 44 out of 47 cases. This was the most striking pathological finding at autopsy. Hemorrhage was seen in the endocardium. In only one case was there a well developed vegetative endocarditis, which occurred in conjunction with multiple infarcts of the kidney and spleen. The kidney showed subcapsular and cortical petechial hemorrhages. Marked acute fatty change was often present in the kidney. In the lower jaw marked hemorrhages were seen beneath the incisor teeth and below the periosteum. One animal developed a suppurative cholecystitis from which a pure culture of Streptococcus viridans could be isolated. As a rule, petechial hemorrhages into the mucosa of the gall-bladder were considered as evidence of gall-bladder involvement. The data obtained, when studied, indicated that the Streptococcus viridans, taken as a group and without differentiation may produce lesions in rabbits. These lesions are identical regardless of the source of the organism used. Those strains from patients without systemic diseases developed in rabbits experimentally by cultures from their abscesses is one the author is unable to answer. He feels, however, that specificity or elective affinity cannot be considered as depending entirely upon the microörganism.

A Cutaneous Reaction in Canine Distemper.—FERRY, MCGOWN, TORREY and RAHE proved that canine distemper is due to a bacterium named by them Bacillus bronchisepticus. KOLMER, HARKINS and REICHEL (*Jour. Immunol.*, 1916, i, 501) prepared a very thick polyvalent emulsion of a seventy-two-hour growth of this organism which they killed by heating to 60° C. for one hour. This emulsion they called bronchisepticin. They used this emulsion in doses of 0.1 c.c. intracutaneously on a large number of dogs. As in other diagnostic skin reactions marked papules, pustules or very marked erythema with

edema were noted as positive reactions. Eye reactions were also tried on the same animals but the results were not satisfactory. Reactions were done on four series of dogs. The first series of nine dogs were suffering from distemper at the time of the test. Of these nine dogs, seven gave a positive and two negative reactions. The second series of twenty dogs were known to have had distemper previous to the tests. Of these twenty dogs, twelve gave positive and eight negative skin reactions. The third series of thirty-seven dogs had no history of infection and it was not known whether they had ever suffered from distemper. Of these animals fourteen gave positive and twenty-three gave negative reactions. The fourth series consisted of eighteen dogs which had been immunized by the use of a vaccine prepared with Bacillus bronchisepticus. Twelve of these eighteen dogs gave a positive and six gave a negative reaction. Some of these dogs had several attacks of distemper and between the attacks the skin test varied and gave no indication as to the dogs' susceptibility. Owing to the inconclusiveness of these figures the authors make no positive claims as to the efficiency of their bronchisepticin antigen as a diagnostic measure, though they believe that owing to the high percentage of positive reactions in dogs having a history of distemper the antigen may be of value. They state, however, that even though it has some value in this way, there is no indication as to the index of the immunity to Bacillus bronchisepticus infections.

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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES

MAY, 1917

ORIGINAL ARTICLES

CHEMOTHERAPY IN TUBERCULOSIS.[1]

By Paul A. Lewis, M.D.,

HENRY PHIPPS INSTITUTE OF THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

In its previous lectures on the general subject of tuberculosis the Harvey Society has been especially fortunate.

Speaking on the subject of tuberculosis in 1906, Theobald Smith said, "The present-day problems in tuberculosis which can be approached by experimental or at least by laboratory methods manifest themselves in three different ways:

"1. In the somewhat chaotic condition of opinion concerning the avenues through which tubercle bacilli gain a foothold in the body.

"2. In the wide divergence of opinion concerning the relation of bovine to human tuberculosis.

"3. In the general trend of studies toward the problem of specific immunity, with especial reference to prevention and treatment."

He presented and critically considered the evidence bearing on the portals of entry of the tubercle bacillus and laid stress on the major significance of the direct infection of the lung and associated lymph nodes by way of the inspired air.

Two years later Calmette presented a great deal of material bearing on the proposition that the tubercle bacillus gains its entry

[1] Lecture delivered before the Harvey Society of New York, November 25, 1916.

similar to the virus, and in stained smears it appears in minute Gram-positive coccus-like bodies arranged in pairs, groups and chains. These minute forms disappear when the organism is cultivated in other media under aerobic conditions. The morphologic, cultural, and pathogenic characters of the poliomyelitis coccus thus far determined indicate that it is an important factor in the disease.

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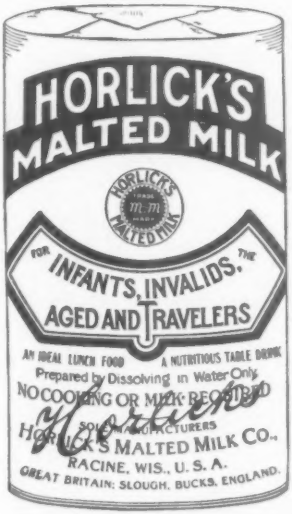

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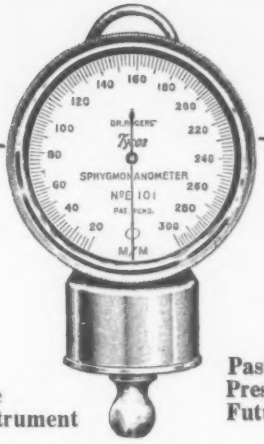

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